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THE FIELD OF VETERINARY MEDICINE

A profession or an individual may be regarded as standing every day at a fork in the road, since each day's selection of alternate lines of conduct more or less determines future developments. From time to time we must look at our road map, ascertain where we are, and decide on our next point of destination. The future is too much of an uncertainty to make a casual progress on just any road a wise procedure.

If there is any sound reason for believing that a veterinarian's training in regard to the diseases of animals makes him superior in his field to other individuals not so trained, then the veterinarian should seriously consider the activities which involve and call for such training, ascertain the extent to which he is involved and employed in those activities, and take the steps necessary to insure that he occupies these fields. Among the steps necessary are the following: The veterinarian must be competent to occupy the field; and he must sell his services on the basis of superior performance.

No small amount of our troubles are of our own making. If physicians were quite generally sound in diagnosis and treatment and their services available at reasonable cost, the physician would have far less competition from the osteopath, chiropractor, mental healer, quack doctor, patent medicine vendor and fakir.

If veterinarians were quite generally sound in the same way, alert to their needs, conscientious about their duties and responsibilities and sagacious in provision for future developments, there would be less home treatment of live stock by the farmer, less activity in veterinary fields by the county agent and others, and less sale for patent medicines and nostrums. While horse practice was good, we neglected other animals. We have since established our practice with cattle and small animals, but still find considerable competition in connection with swine practice, and are still neglecting the possibilities in sheep and poultry practice. Ahead of us lies a promising field of work with captive fur-bearing animals and various wild and zoological park animals.

Consider the fields which we may regard as legitimate activities of the veterinarian. They include the field of practice along the lines of diseases of animals other than man, the field of meat inspection, the field of milk and dairy inspection, and regulatory, research and educational work in veterinary medicine. In some of these fields the veterinarian finds certain other occupants already present. Federal, state, college and university men, including zoologists, bacteriologists and pathologists, are working on diseases of animals other than the domesticated animals. Naturally, their work is primarily a matter of research, but they are supplying educational material to those interested, including the fur-farmer and others. Are we getting our share of this education with the idea that we shall be prepared to step in and take over the control of disease in these animals as a practice? County agents, farm journals and other agencies concern themselves with the treatment of diseases of sheep and poultry in areas where competent veterinarians are not present, as well as in areas where veterinarians are present but do not take an interest in such matters, and sometimes in areas where competent veterinarians are present, who do take an interest in and would like a practice along these lines. Physicians, butchers, market-men and others, innocent of veterinary training, carry on the meat, milk and dairy inspection work for many cities, counties and states.

From this situation we may sort out certain things. There are certain activities, such as research and education, which concern the zoologist, bacteriologist and others as much as they do the veterinarian, and for which activities these men are and will be trained, as well as are veterinarians, and sometimes better. We must cooperate with these men and learn from them. The county

agent should be our ally and cooperator; he should supplement, not supplant, the veterinarian. The business of meat, milk and dairy inspection is one for which only veterinarians are trained, and its importance calls for the use of trained men.

Recognizing that we have made mistakes in the past, we can nevertheless wash these off the slate and focus our attention on the present and future. We must appreciate our needs for training along new lines and better training along old lines. Our college curriculum must be adjusted from time to time to meet new needs. We must foresee new developments in practice and prepare for them. We must meet the county agents in cooperative programs for our mutual interest and for the welfare of the live stock industry. We must show the public and those in authority the danger and unsoundness of meat, milk and dairy inspection by untrained persons.

This is what we must do if we are concerned about our personal status and that of the profession. If we are satisfied with ourselves, and indifferent as to whether veterinary medicine achieves and maintains an independent and dignified status, or becomes an adjunct to human medicine, or suffers some other fate, we can disregard developments which are now taking place and allow events to take their own course. Other interested groups will shape their programs to take advantage of our indolence, inaction and short-sightedness.

M. C. H.

ON THE USE OF VIRUS

Under the caption, "No Danger in Serum Alone," the November 12, 1930, issue of the *Daily Drover's Journal* (Chicago) carried an excellent editorial on the subject of vaccination against hog cholera. Practically all veterinarians will agree with the opinions expressed by the writer of this editorial. For the benefit of those who did not have an opportunity to read the editorial, it is reproduced here in full:

> The problem of hog cholera control has been solved these many years so far as treatment is concerned. Serum alone will give temporary protection, or the proper combination of serum and virus will give permanent protection. When it comes to application of the treatment, however, there is still much controversy, and almost as many rules and regulations as there are hog states. The whole argument hinges on virus, which is cholera itself, and if improperly used loss results and the disease may be spread instead of checked.
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> As a solution there would appear to be merit to the suggestion that virus be kept entirely off farms except as used by experienced, licensed men who know exactly what they are dealing with. And let farmers use

serum as freely as they will. This would do away with any necessity for regulations requiring farmers to go through a lot of red tape in order to get permission to vaccinate their own hogs. They could use the serum alone, get protection for as long a period as it is needed in commercial herds, save themselves some expense and run not the slightest risk of spreading the disease. And if they followed the practice of vaccinating pigs, the cost of protection would be the cheapest form of insurance.

The whole question is subject to more than the usual attention this year because in many sections it has been a bad year for cholera and the worst may by no means be over. Hogs are paying right now, and may pay even better before another year rolls 'round. And not much else is being produced at a profit. Just can't afford to take the chance of having the herd wiped out by this insidious disease, that may actually appear from nowhere over night. Cholera is calamity when it comes, and the only sure protection is vaccination. It simply isn't a good business risk to take the chance of loss when for a small sum per head the pigs can be protected.

In the first place, this is a big country, with lots of hogs, raised and kept under all sorts of conditions. Herds vary in size from several head to several thousand. Nothing further need be said to explain why there is such a multiplicity of rules and regulations for controlling hog cholera. The density of the porcine population in a state like Iowa calls for something radically different from what may be in order for a state like Maryland, with a rather sparse distribution of hogs. It is probable that there always will be considerable variation in the policy of controlling hog cholera to meet the exigencies of local conditions.

To the suggestion that the use of virus be restricted to properly trained and legally authorized persons, all live stock sanitarians will agree without question or quibble. This general policy has been promulgated and endorsed by numerous organizations, interested in animal disease control, time and time again. It is fundamental, and just as long as state laws permit the promiscuous distribution and use of virus, just that long will the swine industry continue to be menaced by the most serious of swine diseases.

It is somewhat of an anomalous situation when we consider that in the double treatment (serum-virus vaccination) we have one of the most satisfactory and effective means of prophylaxis against disease that was never devised, while, at the same time, it involves the use of the full-strength causative agent of the disease—"cholera itself," as the editorial quoted states—for permanent immunity. All efforts to perfect a substitute for the Dorset-Niles-McBryde method, one that would eliminate the use of active hog cholera virus, have signally failed.

The suggestion that farmers should be encouraged to make more general use of the serum-alone treatment is open to question. In many localities it would not be sound practice. If the custom

should become more general, it might result in the use of more serum by farmers themselves, as there would be many instances in which the protection afforded by the single treatment would be of too short duration and the hogs would have to be immunized again, later in the season. With the growth of the hogs, there would be need for an increased dosage of serum, with the incident increase in expense. This added cost would be more, in practically all cases, than the fee of a veterinarian called to vaccinate the herd with serum and virus earlier in the season.

Prophylactic vaccination is rarely an emergency service. Nine times out of ten a herd may be vaccinated just as satisfactorily on Thursday, as on Wednesday, if the veterinarian has other emergency calls on that day. However, with cholera actually in the herd, an entirely different situation is presented, and every hour counts. The delay of a day in starting treatment means dead hogs. As far as the individual hog-owner is concerned, if he is going to have his herd protected at all, his best bet is to have his veterinarian perform this service just as soon as the pigs are old enough to be immunized.

In states and sections of the country where the hog population is not dense and where there is relatively little hog cholera at any time, we approve of the most stringent regulations on the use of virus by anybody and everybody, veterinarians included. Instances in which virus is absolutely essential in these locations are relatively rare and its use should be by official permission only.

MONEY MATTERS

Examination of a preliminary report on the Association's financial condition, as of December 31, 1930, submitted by Treasurer Jacob, permits us to give our members some interesting, up-to-date information on money matters, in advance of the publication of the final report.

First of all, our cash position was improved approximately $1200.00. This means that our total receipts from all sources exceeded our disbursements for all purposes by about this figure. However, closer examination of the report discloses the fact that the strictly A. V. M. A. operations were conducted at a loss of about $2400.000, while the JOURNAL made a profit of about $3600.00.

The A. V. M. A. expenditures averaged $2.81 per member, which was a slight increase over 1929, but less than the cost per

member in both 1928 and 1927. It would appear that the cost of conducting the strictly A. V. M. A. activities had become fairly stabilized, as the average for the past four years is $2.79.

The balance in the A. V. M. A. fund has been decreasing steadily during the past four years at the rate of slightly over $2100.00 per year. The average number of members during this period has been just below 4100. This indicates that the loss per member per year has been about 52 cents.

As has been pointed out at various times, of each five dollars paid as dues, three dollars is credited to the JOURNAL fund to cover the member's subscription. This leaves two dollars for the A. V. M. A. fund. If it has been costing $2.79 per member to conduct the Association's activities, and with an income of only $2.00, it means a loss of 79 cents per member per year. This loss *actually* has been *reduced*, by income from other sources, to 52 cents per member. (This other income has been largely in the form of application fees of new members, interest on bonds and time deposits, and profits from sale of emblems.)

The annual dues certainly should be sufficient to keep the Association self-supporting. Although our primary interest is not to accumulate money, it would be nothing less than just good business for the A. V. M. A. fund to show a small balance at the end of each year, instead of writing it in red, as we have been doing for four years. With that object in view the Executive Board, at the Kansas City meeting, will recommend increasing the annual dues to six dollars. This will give the Association an additional income of about four thousand dollars per year. This will be sufficient to wipe out the deficit of $2400.00 per year and give us an additional $1600 to use as deemed most advisable.

SHOW YOUR COLORS

Approximately two hundred members of the A. V. M. A. are now displaying their membership signs, either on their desks or in prominent places on the walls of their offices.

One member wrote: "You did not boost the dues this year, so here's an extra dollar for a membership sign." Yes, we will send a sign on approval, if you would rather see one before paying for it.

Our supply of automobile emblems was cleaned out about the first of the year, but we ordered 200 more and are now filling orders the same day as received.

APPLICATIONS FOR MEMBERSHIP

(See January, 1931, JOURNAL)

FIRST LISTING

BRINKER, WILLIAM THEODORE R. 2, Box 82, Leetonia, Ohio
D. V. M., Ohio State University, 1930
Vouchers: W. F. Guard and W. R. Krill.

BUFFIN, V. C., CAPT. KENNETH EARL Fort Sam Houston, Texas
D. V. M., George Washington University, 1916
Vouchers: Charles M. Cowherd and John L. Owens.

BUSIC, WILLIAM H. Susanville, Calif.
D. V. M., Ohio State University, 1929
Vouchers: W. L. Curtis and J. P. Bushong.

JONES, EDDELL CHARLES Platte Valley Serum Co., Grand Island, Nebr.
D. V. M., Kansas State Agricultural College, 1916
Vouchers: Bernhard Witt and Floyd Perrin.

KRAKER, LOUIS GEORGE 758 Lincoln Ave., San Rafael, Calif.
D. V. S., San Francisco Veterinary College, 1901
Vouchers: John F. McKenna and O. A. Longley.

MOOTZ, CHARLES E. 1119 N. Pennsylvania Ave., Mason City, Iowa
D. V. M., Cincinnati Veterinary College, 1912
Vouchers: S. L. Ries and H. Preston Hoskins.

WATSON, V. C., LT. EDGERTON LYNN Carlisle Barracks, Carlisle, Pa.
D. V. M., Kansas State Agricultural College, 1930
Vouchers: Geo. H. Koon and Harry E. Van Tuyl.

WELNHOFER, JOHN WILLIAM 1068 Hague St., Saint Paul, Minn.
D. V. M., Iowa State College, 1930
Vouchers: G. E. Totten and M. E. Schwab.

Applications Pending

SECOND LISTING

Apfelberg, Samuel M., 526 Saw Mill River Rd., Yonkers, N. Y.
Caceres, V. C., Capt. Armando Rodriguez, Castillo de la Fuerza, Box 883, Havana, Cuba.
Davis, Charles Robert, Box 125, College Park, Md.
Fernandez y Malberty, Abelardo, General Lee No. 6 A, Marianao, Havana, Cuba.
Griffin, V. C., Capt. Will C., Fort Brown, Texas.
Hoefling, Ray, 709 Euclid St., Austin, Minn.
Jonas, Salo, Rear 83 Broadway, New Haven, Conn.
Kester, Bruce, Vet. Clinic, Ohio State Univ., Columbus, Ohio.
Muxlow, Thomas Jerome, 320 S. 4th Ave., South Saint Paul, Minn.
Riede, Peter H., Mabel, Minn.
Rife, V. C., Capt. George Jacob, Veterinary General Hospital, Manila, P. I.
Taylor, Rex, 2500 16th St., San Francisco, Calif.

The amount which should accompany an application filed this month is $9.58, which covers membership fee and dues to January 1, 1932, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Alabama Veterinary Medical Association and Short Course for Practitioners. Alabama Polytechnic Institute, Auburn, Ala. February 2-7, 1931. Dr. C. A. Cary, Secretary, Alabama Polytechnic Institute, Auburn, Ala.

Connecticut Veterinary Medical Association. Hotel Garde, Hartford, Conn. February 4, 1931. Dr. E. H. Patchen, Secretary, 83 New Haven Ave., Milford, Conn.

Manitoba, Veterinary Association of. Royal Alexander Hotel, Winnipeg, Man. February 4, 1931. Dr. Wm. Hilton, Secretary, 612 McIntyre Block, Winnipeg, Man.

New York City, Veterinary Medical Association of. Academy of Medicine, 5th Ave. and 103rd St., New York, N. Y. February 4, 1931. Dr. John E. Crawford, Secretary, 708 Beach 19th St., Far Rockaway, Long Island, N. Y.

San Diego-Imperial Veterinary Medical Association. San Diego, Calif. February 4, 1931. Dr. A. P. Immenschuh, Secretary, Santee, Calif.

Ohio State University, Short Course in Diseases of Poultry at. Ohio State University, Columbus, Ohio. February 9-13, 1931. Dr. Oscar V. Brumley, Dean, Ohio State University, Columbus, Ohio.

Kansas City Association of Veterinarians. Baltimore Hotel, Kansas City, Mo. February 10, 1931. Dr. H. J. Hearrington, Secretary, 1130 Franklin Ave., Lexington, Mo.

Chicago Veterinary Medical Association. Altantic Hotel, Chicago, Ill. February 10, 1931. Dr. C. L. Miller, Secretary, 508 S. Humphrey Ave., Oak Park, Ill.

Illinois Veterinary Conference, University of. Urbana, Ill. February 10-11, 1931. Dr. Robert Graham, University of Illinois, Urbana, Ill.

Hudson Valley Veterinary Medical Society. Albany, N. Y. February 11, 1931. Dr. J. G. Wills, Secretary, 122 State St., Albany, N. Y.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. February 18, 1931. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles, Calif.

Louisiana State Veterinary Medical Association. Louisiana State University, Baton Rouge, La. February 18-19, 1931. Dr. H. A. Burton, Secretary, Alexandria, La.

Keystone Veterinary Medical Association. Philadelphia, Pa. February 25, 1931. Dr. C. S. Rockwell, 5225 Spruce St., Philadelphia, Pa.

Ohio State University, Conference of Veterinarians at. Ohio State University, Columbus, Ohio. March 18-20, 1931. Dr. Oscar V. Brumley, Dean, Ohio State University, Columbus, Ohio.

DISEASES OF THE MOUTH IN SMALL ANIMALS*

By E. A. EHMER, *Seattle, Wash.*

In presenting this subject, please understand that no attempt has been made to produce a highly scientific paper, but rather to enumerate and briefly discuss most of the important conditions encountered in small-animal practice, in a simple, comprehensive manner. Time does not permit of much detail. However, any operation mentioned herein will be discussed in detail, should it be desired, after the paper is read.

The subject matter will be discussed according to the following outline:

Dentition
Extractions
Stomatitis
The gums
The tongue and sublingual region
Warts in the mouth
Ranula
Necrosis of the jaw
Fractures of the jaw-bones

While these headings are legitimate divisions of the subject proper, it is difficult to present them in a connected manner. Consequently there will be an abrupt change in thought, as one part follows another.

DENTITION

As a rule, puppies at birth have no teeth through the gums. At the third or fourth week, the temporary incisors appear, and are replaced by permanent ones at four or five months. There is no temporary first molar in the dog. The temporary molars appear at five or six weeks, and are replaced at five to seven months. Thus the dog has a full set of permanent teeth at seven months of age. The permanent teeth number twelve incisors, four canines, and twenty-six molars, making forty-two teeth in all.

During the shedding of the temporary teeth and the eruption of the permanent ones, certain difficulties may arise. The temporary incisors usually shed with little or no difficulty but the canines need more careful attention. There is a time when the canines become loosened, and by careful manipulation these can be extracted readily. However, if the proper time elapses, the

*Presented at the sixty-seventh annual meeting of the American Veterinary Medical Association, Los Angeles, Calif., August 26-29, 1930.

teeth seem to take on new life and embed themselves in such a manner that they resemble the permanent ones in stability.

Temporary canines are buried in the gums and jaw-bone about twice the length of the part that is visible. In extracting these, it is seldom possible to remove the entire root. Even in those which seem to loosen themselves and practically come out of their own accord, the exposed portion separates itself from the root and the portion which is shed is hollow at the base. For this reason, should the canines be broken in extracting, it is of little consequence, for it appears to be in accordance with nature that this may happen.

The temporary molars appear as caps which are pushed out by the permanent teeth. These caps are usually displaced by nature, but if not, they are easily removed with the forceps or in most cases can be lifted out with one's finger-nail.

Extractions

Certain breeds of dogs, as the toys, and others, such as Boston Terriers, which have broad and short muzzles, are dental subjects rather early in life. Pomeranians often have misplaced lower canines, which appear in the center of the lower jaw just posterior to the incisors. These teeth grow to a height such that their contact with the gum of the upper jaw pierces it, thus causing constant irritation and a hindrance to mastication.

When both canines are thus misplaced, they usually spread the lower jaw at the symphysis, until the two halves of the mandible can be moved in opposite directions. This is doubtless a reason why dogs of this kind find difficulty in eating hard foods, and it also shows why their teeth are tartar-coated most of the time. Such teeth are more readily extracted than when in the proper location, because of the lightness of the bone in which they are embedded. Nevertheless it is necessary to loosen them well before an attempt at extraction is made, for when once broken off they are very troublesome to remove.

In normal mouths, the first teeth to loosen are apt to be the incisors. As the teeth loosen, a foul breath is noted, caused by food decay and pus at the roots. This process may be retarded by constant cleaning and the application of mercurochrome, tincture of iodin, or hexyl-resorcinol. In many cases the teeth will tighten up in the sockets if the treatment is pushed. However, in the pet dog these loose incisors represent very little value

as compared to the foul breath, and the surest and quickest way to eliminate that is by extraction.

The first and second molars in irregular mouths, such as Boston Terriers, are often placed at such an angle that they grasp and hold food particles, thus causing the formation of tartar, receding of the gums, and finally exposure of the roots. As these teeth are small and are of little value in eating, extraction is indicated as soon as they cause trouble.

As age advances, the other molars often meet the same fate as the first two, and in a few years nearly all of the teeth of some of the toys are lost. This condition can be avoided in a great measure by the avoidance of the softer foods and the feeding of more concentrated foodstuffs which require thorough mastication, which in turn keeps the teeth clean and the gums firm.

Proper extraction of solid teeth is no simple matter. Dogs' teeth are much more brittle than human teeth and often crumble before enough pressure is exerted to loosen the roots. This being the case, it is necessary to find an aid to the forceps. For this, a small chisel and a pair of elevators are most valuable. The chisel is inserted alongside the tooth and forced down into the alveolar cavity slowly, until it is loosened from all sides. Then with the aid of the forceps and an elevator, the tooth is lifted and pried from its socket, with the roots intact. Much time must be spent in the loosening, as when one gets in a hurry, one or more roots are left in the jaw-bone.

Caries and tartar: The tooth of a dog is seldom affected by true decay. The main source of trouble is the formation of tartar, which occurs for the most part on the outside of the upper molars, though all teeth may be affected. This tartar collects in such quantities that it pushes the gums back until the roots are entirely exposed. It is not uncommon to hook a scaler into the tartar and have the entire tooth, roots and all, come out. In such cases, needless to say, the gums are badly inflamed and extremely tender, and the patient eats with the greatest difficulty.

Broken teeth: There is very little clinical evidence that dogs suffer from toothache. Broken teeth are undoubtedly painful for a short time but very soon the tenderness appears to leave. This is borne out by the fact that many dogs are encountered which have broken teeth and show no pain in eating nor flinch when the broken tooth is touched or tapped. It is a common practice among fox-ranchers to cut off the canine teeth of their

vicious foxes and even this seems to be painless within a few days time.

Coating on teeth: In some cases of sickness, mainly gastro-enteritis and distemper, there is produced a material which ranges in color from yellow to dark brown, which is deposited on and strongly adheres to the teeth. By chemical analysis this deposit has been found to contain the elements found in tartar as well as microbes and blood. In the brownish-coated cases we find, besides very offensive putrid breath, a tendency of the gums to bleed easily. Merely touching the gums with moistened cotton will cause hemorrhage. Coated teeth should be cleaned very often with cotton swabs dipped in normal salt solution, followed by the application of a good mouth antiseptic. Persistent treatment will work wonders in such cases.

Distemper teeth: There is a condition in which we find the enamel eroded away in spots, or pits, which in turn become brown. As age advances the entire ends of the teeth erode away until the teeth wear down nearly to the gums. This condition is known among most clinicians as "distemper teeth," as it appears in dogs that have had the disease at four to seven months of age. Some men claim that any severe sickness at this age will produce the same effect on the teeth but the fact remains that one seldom, if ever, sees a case of distemper in a dog with such teeth. In so-called "distemper teeth," the damage is done and there is nothing to do about it. Some of the darkest spots may be cleaned up but for the most part cleaning or other treatment is of no avail.

Stomatitis

Stomatitis is defined as an inflammation of the mouth. We are concerned mostly with necrotic stomatitis. A form of stomatitis which, for the present, will be designated as "acute necrotic stomatitis," is a very virulent type of mouth ulceration which is usually death-producing. First symptoms are tenderness in the mouth, as evidenced by careful chewing, loss of appetite, drooling, and very foul breath. Examination reveals grayish sloughing patches on the tongue, gums, and buccal membranes. These patches peel off until the entire inside of the mouth is devoid of mucous membrane.

The cause of this is unknown to the writer, but appears to be a combination of very virulent organisms which possibly gain entrance to the mouth with decayed matter. Treatment, although usually ineffective, must be drastic, and consists of

swabbing the raw areas with twenty per cent silver nitrate solution once or twice daily, followed by nearly constant irrigation to prevent the absorption of deadly toxins.

Another form of stomatitis appears in advanced cases of gastro-enteritis, typhus and in some cases of distemper. This is evidenced by brown coating of the teeth, very foul breath, inappetence, soreness and drooling. In this form, the necrotic patches are fewer, and less extensive than in that mentioned above, and it appears suddenly, during the general course of the sickness, but responds quite readily, provided the general condition is satisfactory.

Keep the teeth clean, wash the mouth with saturated solution of potassium chlorate, and touch the ulcers with hexyl-resorcinol. Quite satisfactory prophylaxis can be obtained by careful attention to the cleanliness of the mouth during the early stages of the general sickness.

Chronic stomatitis is a condition of ulceration of the sides and end of the tongue, both sides of the gums, and at the angle of the jaws and perhaps extending into the throat. In this form we have the usually offensive breath, drooling, and careful eating, but not the extreme soreness that accompanies the more acute form. The appetite is not lost and while mastication is painful it is carried on sufficiently to nourish the body.

Ten to thirty per cent silver nitrate applied daily to the ulcers is of very great benefit and usually restores the mouth to normal health. In all cases of ulcerative stomatitis, attention should be given to the organs of elimination and something alkaline in nature is of benefit when given internally.

The Gums

In a consideration of the gums we find three conditions of importance: bleeding, ulceration and tumors.

Bleeding gums, as has been mentioned, are seen in conjunction with inflammation of the gastro-intestinal tract.

Ulceration: A form of ulceration of the gums which is a little uncommon was seen in a three-year-old Boston. This dog showed tenderness and redness at the line of the upper incisors. The tissues began to recede and erode, forming a granulated appearing raw surface. There was no bleeding in this case. Treatment with various antiseptics failed to improve matters, and as the teeth were becoming loosened, they were extracted. After this the gums healed over in record time and no more ulceration took

place. After a few months, the molars began to show the same thing. These will likely meet the same fate, but to date nothing has been done with them.

Tumors: Tumors of the gums, while relatively infrequent, are serious because they are usually malignant. They usually appear at the base of the incisors or first molars and grow slowly until they protrude above or overhang the biting surface of the tooth. These vary in size, are usually pedunculated, are red in color and at times are as hard as bone. Surgical removal is the only treatment and may be necessary many times. After a time the tumors may recur so frequently that humane destruction of the patient is merciful.

Tongue and Sublingual Region

Traumatisms: Injuries to the tongue are frequent and are caused by bites, rubber bands, strings attached to food, fishhooks, needles, metal clips and many other things, and regardless of the cause the disturbance rights itself rapidly after removal of the offending object.

Gangrene: Gangrene of the tongue may be considered as primary or secondary. Primary gangrene shows up with no previous warning and no tangible cause. The patient appears in normal health as far as can be seen, but all at once is unable to eat or drink. Examination shows a black patch of dead tissue at the end of the tongue. Often a part of it has already sloughed off leaving a long string of dead tissue which adheres to the back part of the tongue, making its use impossible.

Treatment is surgical and requires the radical removal of all affected tissue. On close inspection one will find that the line between good and bad tissue is irregular. The tendency is to try to save the tongue, and, as a result, too little is removed and necrosis continues. So to be safe, remove enough the first time, which means the removal of all of the darkened tissue and a little of the healthy as well. This operation usually stops the necrosis and the tongue heals over, and unless an enormous amount is lost, no untoward effects are noticed.

Secondary gangrene occurs in conjunction with necrotic stomatitis, which is a complication of gastro-enteritis or typhus. This is treated the same as the above, but the results are more in doubt, due to severity of the constitutional disorder.

Lingual paralysis: Occasionally a dog is encountered that carries the tongue protruding from the side of the mouth. This

indicates that the nerves and muscles on the opposite side are weakened or paralyzed, and the muscles on the good side pull the tongue that way. This seems to be an affection of the small breeds and usually appears in aged animals, so that the disturbance is not of major importance. Treatment is of little or no value.

Edema: As a result of injuries, extensive edema of the loose tissue under the tongue occurs. This is a serious hindrance to mastication and often prevents it entirely. Multiple lancing of this dropsical area allows the free exit of the inclosed serum which brings prompt relief.

Warts in the Mouth

Warts, or papillomata, often occur on the tongue, lips and cheeks. Sometimes they nearly cover the entire inside of the mouth. This may affect an individual dog, or, if in a kennel, may include all the young dogs. For this reason it appears to be of an enzootic nature. Most of our literature advises the use of full doses of Fowler's solution as treatment, which is undoubtedly a very good thing. On the other hand, cases which receive no treatment at all clear up in time, so that the necessity of any treatment might be questioned.

Ranula

Ranula is a term applied to a bleb-like swelling or cyst, inside the mouth and against the tongue. Concerning this condition, Mueller and Glass have this to say:

> It generally occurs under or on the side of the ventral surface of the tongue, is covered with the mucous membrane, and rarely painful to the touch, is smooth, round or thin walled, and more or less cylindrical in shape. Often an animal will become very slow in eating, and if the mouth is examined, we find on one side of the tongue and under it a large sized body, varying from the size of the little finger to a chicken's egg, a fluctuating swelling, generally reddish blue in color, and when opened with a knife it is found to be filled with a thick creamy gluelike liquid. Many theories have been advanced as to the cause of this disease; some consider it to be the formation of an ordinary cyst, and others contend that it is due to the plugging of the ducts of one or more of the salivary glands at the base of the tongue. In some cases the cause of the trouble is due to the obstruction of the duct of Wharton, which has its entrance into the mouth at the base of the lingual ligament, and in other cases it is a cystoid degeneration of a few glands at the base of the tongue, probably due to a plugging of the opening of their ducts, and a consequent inflammation of the glands themselves. It therefore seems best to call all the cystoid formations under the tongue, ranula.

The treatment of ranula is surgical. If located under the tongue or in the anterior portion of the mouth, removal, by very careful dissection, of the entire cyst is advisable. However, if

the swelling extends back into the throat, other procedure is necessary. In this case, lance freely, laying the cyst wide open, remove the contents and cauterize the entire inside of the cystic wall with the actual cautery or other agent such as twenty per cent silver nitrate. This causes sloughing of the membranes, and is followed by considerable swelling and soreness, and inability to eat for a day or two. Irrigation and swabbing of the mouth, several times daily, aids in ridding it of the necrotic tissue and on the third or fourth day, healing is well advanced. In many instances this process must be repeated as the ranula continues to recur. On each successive lancing the membranes are more thickened, and there is apt to be considerable fibrin or fibrin-like material present, besides a quantity of fluid. It is not uncommon to find dozens of tiny calculi inside the ranula.

It so happens that recently we have had a number of cases of ranula to treat. One Boston Terrier had been operated once unsuccessfully a week before it came to us. This one had one small cyst on the left side, which disappeared with one cauterization. On the right side was a larger one, which was operated three times before it stopped forming. In the third operation we were able to dissect it out completely, as it was more anterior in the mouth and this time it failed to return.

A mature German Shepherd was operated and cauterized four times over a period of three months, with apparent success, but after six months it was back again. This time, being well forward, it was extirpated completely and to date has not recurred.

A French Bull, seven years old, was recently encountered with a ranula which extended from the frenum of the tongue to a distance of three inches back of the epiglottis. This one was drained through a small opening, and the entire cavity filled with tincture of iodin, which was allowed to remain for a minute or two and was then syphoned off. For twenty-four hours the patient felt fine and took food. The second day the temperature started up and signs of intoxication appeared. Free irrigation of the cavitywas hindered by the temperament of the dog and a great deal of absorption followed. The temperature continued to rise, followed by weakness, coma and death on the fourth day. Should we meet such a case again, it is our intention to attempt to provide drainage through the anterior portion of the neck, and in this way prevent absorption of the toxins which formed due to the destruction of such a great amount of tissue.

In discussing the subject of ranula with physicians, we learned that it is also very troublesome in the human and repeated operations are usually necessary.

Necrosis of the Jaw

The above mentioned French Bull was troubled by a necrosis of the lower jaw about a year previously. The sixth molar had been extracted three months previously and the cavity failed to heal. The jaw-bone was enlarged to twice the normal size at this point, was very painful, and the socket was deep and necrotic, and had the characteristic odor of necrosing bone. This socket was curetted until reasonably healthy bone was reached and lunar caustic was plentifully applied every third day for four treatments, after which the odor and pain left and healthy granulating tissue appeared, which continued until healing was complete.

Necrosis of this type affects the jaw-bones at almost any point. A middle-aged Boston Terrier had extensive necrosis of the area occupied by the left upper canine and the two teeth on each side of it. The teeth were out and the necrosis had extended into the nasal cavity. This looked like a nearly impossible one but the same treatment as mentioned above was used and the recovery was complete.

Fractures of the Jaw-Bones

Traumatic injuries of the face often involve fractures of both the upper and lower jaws. A blow on the end of the muzzle will frequently fracture one side of the upper maxilla in such a manner that the muzzle is turned slightly to one side, and it is impossible to close the jaws. This is a very peculiar sight and at first glance one is apt to believe the lower jaw is the one out of place. Closer observation will show a crushing of the upper maxilla, possibly with a dislodged molar tooth. Should this happen to a long-muzzled dog, it can be treated successfully by pulling the maxilla into proper line and applying a strap muzzle around the snout sufficiently tight so that the canine teeth will remain locked and thus hold the jaws in place. Healing is very rapid.

Fractures of the lower jaw are much more frequent. Symphyseal fractures can be handled nicely by placing a wire around the canine teeth and twisting it tight.

Fracture of one side of the jaw, anywhere between the canine and last molar, is often treated by the application of a wire

around the teeth just mentioned, and twisting it tight. When possible, a muzzle is a big help in this case. The wire usually comes off two or more times during the process of healing but another can be placed readily. It is not necessary to keep the wire on until healing is complete. As soon as the skin is healed and there is a little stability in the jaw itself, further artificial help is not needed.

Bilateral fracture of the lower jaw requires more serious consideration. When this happens, the jaw hangs limp, closure being impossible. Such cases often look hopeless and it is often with difficulty that an owner can be convinced that there is a chance for recovery. If properly handled, these cases do very well. To treat this, drill through the jaw-bone about three-eighths of an inch to each side of the fracture, going to one side of or between the roots of the tooth, which may be at that point. Pass a twenty-gauge silver wire through the drill-holes and double it, then twist it up tight, cut the ends as close as prudent, and bend the twisted ends down flat against the gums. This wire imbeds itself into the tissues very quickly and in a few days is nearly out of sight. The patient is at first very timid about eating but in a day or two will lap liquids and even attempt soft food.

The odor from the mouth is very offensive and there is considerable drooling at first. After two weeks this lessens. If the patient will allow it, frequent washing of the mouth, by squirting water into it with a syringe, will reduce the bad odor and materially hasten healing.

There are two points concerning which one should be cautious. Do not make the holes too close to the broken ends. If this happens, the small area is apt to break off, or necrose and slough off. In either case the wire support is of no value and more drilling and wiring must be done. Another point to watch is the twisting of the wire. The tendency, when tightening is under way, is to give it just one more twist and if it is one too many, the wire will break and the process of threading the wire through the drill-holes must be done over.

Some of these heal faster than others, so it requires judgment to know when they have worn the wires long enough. However, it usually happens that the wires become loosened after a time and after three or four weeks you will find that they are not holding much. At this stage it is alright to remove them.

The time required for complete healing varies from one to six months. This must be considered before treatment is com-

menced. One little Boston Terrier received a blow from a car which fractured the lower jaw an inch below the articulation. This one was impossible to reach from inside the mouth so the skin and muscle were laid open on the outer side and silver wire used to hold the bones in apposition. This dog, contrary to our expectations, had no difficulty whatever with the muscle and skin wound. It was feared that the saliva from the inside would seep through and cause us trouble but that did not happen. In fact, the wound healed by first intention. The bone, however, did not do so well. More than a year has elapsed and there is still crepitation, though the dog eats fairly well. It appears to be a matter of extremely slow healing, as the x-ray shows nothing else wrong.

There may be a tendency to give up on some of the more severely fractured jaw cases. Do not do this if there is even a slight chance, for one never knows what can be done until he tries. Many times the fee will be small and much time has to be spent, but a case of this nature which terminated with recovery is broadcast far and wide and the favorable mention which one gets is worth more than a big fee would have been.

In closing may I be permitted to digress from my subject long enough to make a plea for better service, better knowledge, better surgery and the improvement of everything which is at our command, which will give us prestige and place us on a higher plane among the learned professions. Some are inclined to take advantage of the fact that the dog's abdomen will stand a lot of mauling and infection and consequently do surgery in a haphazard manner. This is not so bad if done in private but when the public is invited to look on, it certainly does not add to the dignity of our profession. This has been inherited from those of the old school, and is on the decline, but there is a world of room for improvement.

Careful surgery is no more expensive nor time-consuming than the other variety and the satisfaction of knowing that things are right is inspiring, to say nothing of the much better results one obtains directly with the patient.

DISCUSSION

DR. J. M. ARBURUA: I am very much interested in a subject that Dr. Ehmer did not touch on. I am wondering what experience he has had in sewing slight harelips in new-born puppies, and not cleft palates?

DR. E. A. EHMER: A few years ago we attempted to correct a few cases, but we didn't have very good results and I haven't attempted much in that line since.

DR. J. B. HARRISON: I have tried suturing quite a number of harelips, and have kept on experimenting with them until I figured out a sort of subcutaneous cross-stitch. It is hard to describe. I use a stitch in the skin—and it is subcutaneous, or invisible, when you get through—and tie it away up in the nose so they cannot get at it. First I tried suturing them through and they took those out, but I have obtained very nice results by using this subcutaneous stitch and tying it clear up in the nose where they cannot reach it.

DR. W. L. CURTIS: I would like to ask the last speaker what age most of the pups are when he does the operation?

DR. HARRISON: You have to wait until they are around three, four or six months old. I had one Pekingese that was six months old and made a very nice job of it. You have to cut the lip loose, on the side where it is attached, to get enough skin to pull in, and it gives very nice results. If they are bad you will have to suture them on the inside first before stitching them together on the oustide.

DR. ARBURUA: Don't you think it should be advised in new-born pups?

DR. HARRISON: I don't think you can do it then, because the skin is not thick enough to hold your suture, even though you use the finest of gut. You may be able to put in a plain interrupted suture there, and get it, but I think you get better results by waiting until you get a little skin to work on. I have never tried it on new-born pups, but some that I tried it on were about two months old and I did not have much luck and would advise waiting until they get a little older.

DR CURTIS: What percentage of the pups die?

DR. HARRISON: If they have a bad harelip, practically all of them die. Where you get a real bad harelip, you have a cleft palate in most every case, but I find a few cases where you have a harelip without a cleft palate.

DR. G. W. FOELSCHOW: In reference to a dog with warts, supposing you have the mouth, cheeks and tongue full of them, what would be your technic when your client insists that you do something to remove them?

DR. EHMER: I used to listen to them when they insisted on that, but I do not any more. I do not think you should. I think you will make a sore mouth worse. They appear to be, without question, infectious and as soon as you get one out you will have a new crop. Of course, if you take the membrane out with the wart, you can get away with that, but those things disappear so mysteriously that it is hardly worth while I believe, to interfere with them surgically.

DR. FOELSCHOW: I happened to have that unfortunate experience, although it turned out very fortunately. I found a mouth just completely covered with warts and it was impossible to dissect them. So I painted the mouth and removed them practically all by scraping them off. It was marvelous the recovery that followed. As you say, possibly they would have disappeared anyway. It pleased my client.

DR. J. C. FLYNN: I find that where you get a client who thinks that something must be done, and you don't want to resort to surgery—I think it is a bad idea in those cases—I frequently put them on arsenious acid, 1-60th of a grain three times a day, and tell them to swab the mouth frequently with castor oil. That softens up those little warts and it is only a matter of a short time until they disappear. Where they are so numerous that there is necrosis from the sloughing over or crowding of the tumors, it is possible, if of a serious enough nature, and you have attempted to remove them surgically, to get into serious trouble and cause the death of your animal more quickly than if you let him alone. Some of them will die from absorption and some from absolute strangulation, where the tumors are numerous and large enough.

A rabies quarantine was placed on Jefferson County, Illinois, during December, as the result of a fresh outbreak of rabies in the County. This was the second rabies quarantine for Jefferson County during the past year.

THE DETECTION OF HORSE MEAT AS AN ADULTERANT IN SAUSAGE, AND OTHER STUDIES OF THE PRECIPITIN TEST*

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Introduction

The increasing importance in the horse-meat industry, and the relationship between laws, public demand and competition have stimulated the preparation of this paper. A laboratory verification of meat adulteration is frequently requested by meat inspectors, when men in charge of meat inspection notice firms selling a meat product for less than actual cost. Many horses are slaughtered each year for foreign trade and for domestic consumption. Some firms have put horse meat in hamburger and sold it in competition with the beef products. Then, too, the State Game Commission has rare species of animals which have no open season or only a short season in which hunting is permitted. To some people this increases the value of this flesh as food. When the game wardens receive a tip that an elk or deer has been killed, they visit the place and find that it has been canned, pickled, or dried. Such material is sometimes brought to the laboratory for identification. This paper gives the results of attempts at identifying such specimens. It is, however, only a preliminary report.

The precipitin test is the one which has been used. It consists of a process of immunization of a suitable animal against a specific protein. The animals immunized were a calf, rabbits and guinea pigs. Proteins from closely related species and age of the animal immunized were factors entering into the reactions. This test is a very delicate one and therefore errors are easy to make.

For convenience a detailed description for the detection of horse-meat adulteration in sausage, followed by a discussion of cooked-meat adulteration and elk-meat identification, will be given.

Test for Raw Horse Meat

The test is divided into three parts: (1) Preparation of the

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anti-serum; (2) preparation of the suspected meat extract; (3) the test proper.

Preparation of the antiserum: The preparation of a specific immune serum begins with the preparation of a specific protein for injection purposes. If a fresh meat is to be examined, it seems preferable to make a meat extract using an .85 per cent solution of NaCl rather than the serum of the meat to be determined. When a meat extract was set up against a serum-immune serum of the same species, the reaction was delayed fifteen or more minutes. It was also very slight, and was therefore confusing. Fresh extracts should be made each time they are to be used. The injection of a contaminated extract will result in the production of a precipitin for the organism, and result in error of the interpretation of the test. Also the injection of a contaminated extract will result in the death of a few animals from dysentery and pneumonia.

Injections may be intravenous, intraperitoneal, subcutaneous or subarachnoid.[1] Subarachnoid injections have not been so satisfactory, mechanical injury being the cause of death. Intravenous injections, at three-day intervals, of 5, 10 and 15 cc for a five-pound rabbit, or less for younger rabbits according to size, are very satisfactory. Several rabbits should be used for each protein to be tested, as every rabbit does not produce satisfactory immune serum. The vein along the side of the ear is the best one to use.

A 22- or 24-gauge needle and a glass syringe were used to make injections. Ten days to two weeks after the last injection, the animals are bled. They should be starved out 24 hours before bleeding to avoid the minute fat globules in the blood, which make the serum opalescent and interfere with the reading of the test.[2] Blood is taken from the heart while the rabbit is under ether anesthesia. The blood is then centrifuged and the serum transferred to another test-tube. Often times the corpuscles are thrown down and leave the fibrin which coagulates and takes on the appearance of gel. The serum clot can be loosened from the test-tube and thrown down when centrifuged the second time. In these tests, rabbits retained a very high titre for two or three weeks, after which it began to fall. An injection at this time will bring the titre back to a satisfactory level. The procedure should be to give from 0.5 to 1.0 cc of diluted extract, followed in an hour or so with 2.0 or 3.0 cc of the meat extract, given intravenously.

Fatalities from anaphylactic shock in these rabbits are very high. Artificial respiration, by forcing the air out of the lungs, should be tried, as well as the administration of adrenalin in small repeated doses, which is recommended by some workers. Smith[3] cited Auer as having said 3 mg. of atropin injected into the subcutaneous tissue of sensitized guinea pigs before reinjection of the antigen protected three out of five against immediate death. Smith[3] also made reference to Banzhof and Famulener, who found that 100 to 150 mg. of chloral injected intramuscularly into sensitized guinea pigs saved from 90 to 100 per cent of the animals from anaphylactic shock.

The preparation of the unknown extracts: Meats to be used should be taken from the center of the specimens, to avoid contamination with other meats. A gram or two is placed in a beaker, covered with .85 per cent sodium chlorid solution, and incubated for 30 minutes at 37° C. It is then filtered through paper or centrifuged to remove the particles not in solution. The dilutions may be made by comparison with the bubbles formed when air is forced through the protein solution, as compared with a known dilution of serum, preferably 1:1000.

The test: The titre of the immune serum is established against the specific protein used for the immunization of the rabbit. Dilutions of 1:100, 1:500, 1:1000, 1:5000, 1:10,000, 1:20,000, and 1:40,000 are made.

Test-tubes 8 millimeters in diameter are satisfactory. Two tenths cc of the different dilutions are placed in tubes. A 1-cc pipette may be used to add the immune serum; starting in with the greatest dilution, lower the delivery end of the pipette to the bottom of the liquid. Slowly allow 0.1 cc or a sufficient amount of serum to escape to bring the serum above the curve in the bottom of the tube, and a sharp line of contact will be seen between the extract and serum. Withdraw the pipette, so as not to agitate the serum. Continue from the highest to the lowest of the dilutions in like manner. In reading the test, a shaded light is desired. A north window is best. The rack is placed so that the light falls on the line of contact between the immune serum and dilution at an angle. With the eyes on a level with the line of contact, looking towards a dark background, a white clouded ring can be seen in a very few minutes if the test is positive. A separate pipette should be used for each solution after the titre is established. A 0.2-cc pipette is large enough for this.

TABLE I—*Rabbits immunized by systematic injections of horse serum*

ANTIGEN	SERUM	REACTION
Horse serum (1:1000)	Horse-serum-immune rabbit	+
Bovine serum (1:1000)		+
Horse-meat extract (1:1000)		—
Beef extract (1:1000)		—
.85 NaCl		—
Suspect		—
Suspect	Non-immune rabbit	—

The horse-serum-immune rabbit serum had a titre of 1:10,000. Reactions occurred in both horse and bovine serum at a dilution of 1:1000, while the horse-meat extract, at a dilution of approximately 1:1000, did not react in 20 minutes.

TABLE II—*Rabbits immunized by systematic injection of horse-meat extract*

ANTIGEN	SERUM	REACTION
Suspect	Horse-meat-extract-immune rabbit	+
Horse-meat extract		+
Beef extract		—
Mutton extract		—
.85 NaCl		—
Suspect	Non-immune rabbit	—

Horse-meat-extract-immune rabbit serum gave positive reactions with the horse-meat-extract dilution (approximately 1:1000) in three minutes, the suspect in four minutes, and did not react with the beef or mutton extracts at the end of twenty minutes.

TEST FOR COOKED HORSE MEAT

Rabbits and guinea pigs shown in table III were immunized with a cooked product. The method used is a modification of the method used by Volta and Carpio.[4]

Cooked horse-meat antigen: Ten pounds of horse meat was ground finely in the food-chopper. To this product 5 pounds of distilled water was added and it was placed in the ice-box over night. The liquid portion was strained through cheese-cloth in 24 hours. The material was then placed in the ice-box until the following morning, when the solution on top was siphoned off, leaving the sediment. Five hundred cc was obtained, which compared to a 1:10 solution of serum and of water.

To the 500 cc of horse meat extract, 25 grams of ammonium

sulfate and 30 m. of acetic acid were added. The material was then heated in the water-bath until no more precipitate formed. The heated product was then filtered through paper. The material remaining on the filter was placed in the incubator at 37° C. to dry out. The dried material was kept in the refrigerator at 0° C. When ready to be used, the antigen was ground in a mortar very finely and put into an emulsion in warm .85% NaCl solution. The animals were injected every 2 or 3 days with 2 or 3 cc of the antigen. There were 4 or 5 injections made. The animals were bled from the tenth to the twelfth day following the last injection.

Preparation of the extract for dilution: Extracts 1 and 2 were prepared by taking a sample of ground meat, placing it in a beaker and covering with .85 per cent NaCl solution. This material was placed in a water-bath and boiled for 30 minutes.

Extracts 3 and 4 were prepared by taking a block of the meat, putting it into a beaker and heating in the autoclave at 15 pounds pressure for 10 minutes.

The fresh meat extracts were made by the physiological salt solution and incubation method.

TABLE III—*Tests for cooked horse meat*

EXTRACT	COOKED-HORSE-MEAT-IMMUNE SERUM	NON-IMMUNE SERUM
(1) Horse meat heated to 212° F.	+	—
(2) Beef heated to 212° F.	+	—
(3) Horse meat heated in autoclave at 15 lbs. for 10 min.	—	—
(4) Beef meat heated in autoclave at 15 lbs. for 10 min.	—	—
(5) Horse meat extract not heated (approximately 1:1000)	+	—
(6) Horse serum (1:1000)	—	—
(7) Beef extract (approximately 1:1000)	—	—

Because the horse meat heated in the autoclave did not react with the immune serum (table III), a series of five rabbits were injected with the liquid left when horse meat was heated in the autoclave. Three of the rabbits died with a dysentery. It was due to the toxic effect of the cooked product. The other two withstood the injection of 5, 10 and 15 cc at three-day intervals. They were bled on the twelfth day following the last injection. There were no precipitins present for the specific antigen or other proteins.

The Test for Elk (Cervus Canadensis) Meat

A series of rabbits were injected with elk meat extract. The results obtained from this series are shown in table IV.

Table IV—*Test for elk meat*

Antigen	Serum	Reaction
Elk-meat extract	Elk-meat-extract-immune rabbit	+
Beef extract		+
Horse-meat extract		—
Suspected extract		+
Suspected extract	Non-immune rabbit	—
Elk-meat extract		—

To get away from the closely related species reaction, a four-weeks-old calf was chosen as the animal to be immunized. The calf was given 10 cc of elk-meat extract into the subcutaneous tissue. The first injection was followed at three-day intervals with 10, 20 and 40 cc of the elk-meat extract given intravenously. On the twelfth day following the last injection, the calf was bled. The results obtained from this process are shown in table V.

Table V—*Test for elk meat with calf serum*

Antigen	Serum	Reaction
Beef-meat extract	Calf immunized to elk-meat extract	+
Elk-meat extract		—
Elk serum		—
Beef serum		+

With the above results in mind a reference was found, which has since lost identity, that precipitins are found in the blood of the calf for that of the dam and likewise precipitins in the blood of the dam for the calf. This is not true in all cases, however.

Two series of rabbits were then injected with meat extracts, one for elk, the other for beef. The test was then run, one against the other. By making a series of dilutions and using one from each series that reacted in the same manner for the specific protein, it was possible to cut out the other for a short while. With this combination the suspected meat was established as being not elk meat but as being beef. An extract of the suspect reacted much sooner and greater with the beef-immune rabbit serum than did it with the elk-immune rabbit serum.

TABLE VI—*Tests with immune rabbit sera*

EXTRACT	BEEF-EXTRACT-IMMUNE RABBIT SERUM	ELK-MEAT-EXTRACT-IMMUNE RABBIT SERUM	NORMAL RABBIT SERUM
Beef (approx. 1:1000)	+ in 5 min.	— for 5 min.	—
Elk meat (approx. 1:1000)	— for 5 min.	+ in 5 min.	—
Suspected meat (approx. 1:1000)	+ in 5 min.	— for 5 min.	—

SUMMARY

1. A horse-meat-extract antiserum was better than horse-serum antiserum for the detection of horse-meat adulteration in sausage.

2. Rabbits immunized to a cooked product had specific precipitins for the non-cooked specific protein, and at the same time had non-specific precipitins for cooked meats of different species.

3. Meats heated in the autoclave at 15 pounds pressure for 10 minutes did not make a suitable antigen for immunization purposes.

4. Immature animals were unsatisfactory in the production of precipitins.

5. Meats of closely related species were differentiated only by the length of time necessary for reactions to occur.

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DISCUSSION

CHAIRMAN BUTLER: Dr. Bolin, we want to thank you for that very instructive paper. The paper at this time is of great value to the different states, especially to sanitary officials. I don't know of any papers dealing with the detection of these adulterants that we may find in sausages. Now that we are killing so many horses for slaughter, it necessitates a knowledge of how to detect horse and other meat adulterants.

There are a lot of horses being killed today. They are supposed to kill them for dog meat, but I know in our own county there is many a picnic where you think you are eating beef whereas you are eating horse meat.

So, Dr. Bolin, we appreciate very much your instructive paper. This paper is now open for discussion.

LT. MAURICE W. HALE: Can you identify meats after they have been cooked?

DR. BOLIN: So far we haven't been able to identify them after thay have been cooked. Some of the foreign publications indicate very good results.

Some of our results are encouraging. There are many factors that enter into a cooked product. It may have some basic product added to it, which will make it slightly alkaline, or some acid added to it will alter the preparation in such a manner that it is very difficult to test it. It is very unsatisfactory to make a dilution by the comparison method, especially with the cooked product,

for the simple reason that one can add an alkali and make it bubble very profusely and if slightly acid it won't bubble at all.

DR. W. H. LYTLE: Pardon me for speaking from the rostrum, but I want to give you some idea why we are particularly interested in this paper. We have a horse-killing plant in Portland and it had been the practice in previous years to dispose of the aged dairy cows and old bulls through the hamburger route.

It so happened that the market for aged dairy cows slackened down somewhat. Some of the people who specialized in those animals were wondering about the cause. They reported it was the practice to use the bones that would come from bulls and dairy cows worked up into hamburger to sell to the restaurants for soup-making purposes. Some of the restaurants that attempted to buy those bones told me they could not be had, so that led us to believe that dairy cows and bulls were not being used for hamburger purposes.

We gathered statistics. We found that the large packing-houses were selling only half the number they sold in previous years, and for that reason we asked the College to make an investigation and see just what could be done in working out a test. We had positive reports. In several instances we promptly cancelled the licenses of the markets that were selling this product.

When we gathered statistics we found that last year there were slaughtered, under inspection in the United States, 136,000 horses. That was a fifteen per cent increase over the pervious year.

One of the newspapers from Iowa wrote me a letter making certain inquiries as to the test, and they reported 30,000 Iowa horses had been sent to Rockford, Illinois, last year and some of their cattle men wanted to know what had become of some of that horse meat.

I feel that much of this horse meat has been smuggled from one state to another. We find it is the practice in Portland to grind up certain parts of this meat, place it in a container, a fifty-pound container, and of course that is presumed to be used for fox-feeding purposes, but we know all of those packed products are always found in markets that do a sort of questionable business. I am sure it is an unfair competition and I believe we will have to ask the federal government to make a more stringent regulation as to the branding of horse meat.

Of course you understand that horse meat is permitted to be sold as horse meat, but when it is sold for beef or mutton or as an adulterant, that is unfair.

CHAIRMAN BUTLER: There is a lot of horse meat being used. We had a man arrested for stealing cattle. It became a question of identification of brands and it was quite a complicated case. That man had been operating for twenty years and we had never been able to catch him. I had quite a little to do with the identification of the brands because he would dissect the brand out and sew the skin together and rebrand the animal.

In this particular case we had to go to the slaughter-house out in the country and we were dumbfounded to find about seventy-five per cent of the meat in this house was horse meat. They were selling it as beef.

This is a very important paper and a splendid contribution to the library of anyone who is interested in this particular line of work.

MEMBER: Is it permissible to use horse meat in connection with any other meat food products in sausage without labeling it or by labeling it as such. I mean to use part beef and part horse meat in sausage.

DR. LYTLE: It is purely a legal question, and I imagine that would depend very much on the food laws of the particular state where the condition exists. I know with us we have no laws that adequately deal with this subject. We had our attorney general make an investigation and he reported that in all probability the use of horse meat in hamburger was adulteration, because it was an inferior product, and his definition according to most food laws would constitute adulteration. Substituting a product that is presumably inferior is adulteration. However, much depends upon the laws of the state.

It is reported that Turkey has only one civilian veterinarian, all others being in the military service. The value of the live stock industry in Turkey is estimated at $300,000,000.

THE PART THE PRACTITIONER PLAYS IN THE PROGRAM FOR THE ERADICATION OF BANG ABORTION DISEASE*

By W. H. HENDRICKS, *State Veterinarian,*
Salt Lake City, Utah

In presenting this short paper, I desire to call attention to the fact that I am merely presenting one angle of the problem of controlling Bang abortion disease as I meet it in my capacity as State Live Stock Sanitarian. There are, of course, a number of angles, each probably as important as the others. A great deal has been written, a great deal of experimental work has been done and much more will be done on this disease. Until we develop a definite, uniform and feasible method of controlling the disease, such as has been developed in the case of hog cholera, anthrax, tuberculosis and other diseases, there will be more or less controversy, variance of opinions, and conflicting data such as exist at the present time. Some advocate one plan, some another. Some advocate the use of vaccines and bacterins, others condemn them. It is not my purpose in this paper to discuss these phases of the subject. A number of prominent men are devoting their time to this and other phases and I think that eventually from all the work that is being done a correct and feasible plan of control will be worked out. We are very much indebted to the research workers who are devoting their time to this problem and are continually enlightening us on the subject. We are anxiously waiting for them to solve the problem, but in the meantime we have to meet the situation as it is. The disease is probably the most serious from the standpoint of investment in the cattle industry that is affecting our live stock today.

The disease is rather unusual from the standpoint of eradication and control, first, from the fact that the percentage of infection has already reached large proportions before any definite plan of control has been devised. At least this is the case in our state, and I think it holds true in a great many other states; and so the method of disposing directly of all infected animals, as disclosed by the agglutination test, is quite out of the question. Our present laws, our present legislative appropriations, and the public sentiment certainly would not permit of this method at

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the present time. Second, the diseases that are associated with Bang abortion disease, or as its sequelae, are quite often handled and treated as distinct diseases, separate and apart from abortion disease. For these reasons it seems that for the present, anyway, the disease will have to be attacked a little differently than other diseases have been, particularly from the standpoint of state control and eradication.

State and federal live stock sanitary forces have adopted regulations and plans of control for various live stock diseases. Most of them have been successful, but the nature of the diseases has been such that they caused great losses of live stock through death, or else the financial and public health angle was of such magnitude that it was not so difficult to mould public opinion in favor of a drastic plan of control, which in some cases involved the slaughter and destruction of all diseased and exposed animals, while in other cases we were able to control the disease by the use of biologics.

Seriousness Slow to Be Recognized

With Bang abortion disease the animal is not visibly sick to the average man. The infected cow very rarely dies from the disease. The average cattle-owner has been slow to recognize the consequences of the disease. Rather he has been prone to call upon his local veterinarian to assist him when his animals have been afflicted with what he supposed were separate and distinct diseases but which were really conditions associated with abortion disease.

It is necessary to have the stock-owner informed in regard to these facts, and the practitioner is the man who is qualified and in a position to do this. By this, I mean that the practitioners, located as they are in and among the various cattle sections of our commonwealths, are the men upon whom we will have to depend. They are the men who, by reason of their training and their direct contact with the disease, will probably do the greater part of the job of actually controlling the disease when almost any plan is put into operation. I think, perhaps, there is no other disease in the cattle sections to which practitioners are called upon to devote more time at the present than Bang abortion disease and its sequelae. The research worker, the laboratory man, the live stock sanitarian, all must have the assistance and the cooperation of the practitioner in order to make any definite headway in a plan of control. And the practitioner must in turn

familiarize himself with the disease from all angles, and so inspire public confidence that he can gain the cooperation of the cattle-owners, which is very necessary in the control of this disease. It falls to the lot of the practitioner to treat cases of sterility, calf scours, udder troubles, retained placenta, etc., which brings him constantly in direct contact with Bang abortion disease. The cattle-owners look to him and depend upon him for advice and assistance. In view of these facts, he is in a position to assist very materially in controlling this disease.

The nature of the disease is such that under our present knowledge of control it would not be practicable to ask all stock-owners to practice a single uniform plan, at least, not a detailed plan. The same general plan may be used as a working basis, but within the general plan will have to be provision for exceptions. We have cattle-owners with small herds and some with large herds. These owners usually operate under very different conditions and circumstances, so that a method of handling a herd that would be practicable for one man would be very impracticable or probably prohibitive for another man.

Different Plan of Control Necessary

Due to the percentage of infection, I doubt very much if any of the states are in a position to handle this disease on a plan similar to our tuberculosis eradication, and I do not know that such a plan would be advisable. We may find that animals that have had the disease are probably the most valuable animals to retain. We, at least, may have to change some of our present ideas in regard to controlling the disease. I know that in our state, where agglutination tests, as far as we have gone, are showing a rather high percentage of infection, with the possibility of a number of our negative animals turning positive on a later test, we cannot hope to force all of our cattle-owners to dispose of their reacting animals, nor to maintain two herds, because they are not in a position to do so.

We are operating under a plan wherein we depend largely upon the assistance of the practitioners. Our legislature recently provided us with funds to equip an up-to-date laboratory, and outside of the laboratory assistance, our plan incurs very little expense to the State. As a matter of fact, if it did we probably would not be doing anything. But we are working on the assumption that the cattle-owners have the disease to contend with. It is their problem as much or probably more so than ours, and

so we must have their assistance and cooperation in controlling it. They must bear the greater part of the expense of the work because they are the ones who are directly benefited. They must be educated to the fact that their voluntary cooperation is necessary. This cannot be so easily done if we do all the work free of charge to them. It seems that they will not readily appreciate the results and advantages obtained unless they are put to some expense and trouble. And then, too, the eradication of the disease is more or less an individual problem for each cattle-owner. He is the man directly in charge of the herd and who must see that the details of the plan are carried out.

Therefore, we are enlisting the support of the county agents, who are in direct contact with the farmers all the time, to help us sell the idea of eradication and control to the stock-owners. Our plan in general is explained to them and they are assisting us a great deal by inducing the cattle-owners to cooperate on our plan of control.

Progressive Dairymen Take the Lead

We endeavor to start blood-testing among the herd improvement and bull associations first, because they are usually a class of more progressive dairymen and consequently better cooperators. After a few herds in each section are established as clean herds by reason of a plan adopted which suits their convenience, the results obtained are automatically sold to neighboring herd-owners and the interest in the work gained in this manner makes it much easier to carry out our plans of control. The herd-owner is advised to call in his local veterinarian and consult him in regard to the disease and then to have the veterinarian take blood samples from his herd and submit them to the laboratory for test. When the practitioner receives the laboratory report on the herd, he again visits the herd and explains the test report to the owner and advises him in regard to a plan of control, after having received first-hand information regarding his problems and thereby assisting him to put into operation a plan which best suits his conditions. This work is done at the expense of the owner and gives us, through the practitioners, an opportunity to contact each herd-owner and help him solve his problems. If the herd-owner needs more assistance from time to time, which he usually does, the local veterinarian is always in a position to help him out.

We feel that this work must be more or less uniform. It would not be well to have one veterinarian advising one method and the next veterinarian advising a different method on the same herd, or perhaps herds handled under similar conditions and that should be following the same plan of control; and so each practitioner receives a uniform plan from the State Department which embodies four different methods of handling herds according to conditions met. The veterinarian is in close touch with these herds. He has a chance to watch the results of his work and to profit by the same, both financially and professionally. The practitioner, therefore, plays a very important part in a plan for the control of this disease. In fact, we are depending upon him almost entirely to do the greater part of the work and a very vital part of it. It, therefore, behooves the practitioner to keep abreast of the times and be prepared at all times to impart up-to-date, efficient information to stock-owners and to be in a position to fit into the various plans of control. No one is in a better position to do this than is the practitioner. There are a lot of little details in regard to herd management, sanitation, etc., that the practitioner must impart to the cattle-owners.

The following is, in brief, a general plan under which our practitioners work:

> We feel that it is imperative that all veterinarians be uniform in the plans of control advocated, and so the Department is setting up the following plans which we feel will fit all conditions, and urge that the veterinarians advise handling the herds accordingly. When you get your laboratory report, visit the herd at your convenience and interpret the results to the owner. Then advise and persuade him, if possible, to adopt the plan of control which fits his *conditions*.
>
> *Plan 1. An average herd showing only one to two or three positives.*
>
> In this case the owner should be induced to dispose of the reactors, clean up and strive for a clean herd by repeated testing, careful sanitation, maintaining a maternity stall for parturient animals, and isolating and testing new animals before they are added to the herd.
>
> Reacting animals may be sold into infected herds, but in all cases the seller must advise the purchaser that the animal is a reactor, or he may be the recipient of a just complaint by the purchaser.
>
> *Plan 2. An average herd wherein the reactors range from 20 to 50 per cent or higher.*
>
> This man has a chance to follow one of two plans in addition to plan 1, if he chooses to adopt it.
>
> (1) If he is so situated, we advise the maintenance of two herds, an infected herd and a clean herd, with repeated testing and the possibility of eventually arriving at one clean herd. The points mentioned in plan 1, of course, would apply to this plan.
>
> (2) He may feel that the best he can do is maintain an infected herd, which he desires to do with as much profit as possible. It may be advisable to advocate vaccination in this man's herd. The Department feels that vaccination should be recommended only in exceptional cases. We

should avoid vaccination as much as possible. The owner must be given to understand what it means to vaccinate and maintain an infected herd. Animals will be barred from interstate shipment, especially if an agglutination test is required. It will be more difficult to dispose of surplus animals. Then there is the undulant fever connection and what it may mean from a public health standpoint, by way of regulating the milk supply, etc.

If, after talking with the owner, the veterinarian decides to vaccinate, we request that he advise this office in regard to the vaccination in question, so that the records kept in this office will show the herd to be maintained as an infected and vaccinated herd.

Plan 3. A herd in which the test shows no infection.

In this case the owner will be advised how to maintain a clean herd. In all cases where clean herds are maintained, the herd should be tested twice every year and perhaps oftener. The points mentioned in plan 1 should be particularly stressed in this herd.

Plan 4. A small herd with some degree of infection.

If the owner is not able to follow plan 1, then he may have to follow the second part of plan 2, with the idea always in view of doing as little vaccination as possible.

If we can make a start and everyone be uniform in following one of the above plans according to conditions met, it will do two things. First, it will provide this office with records of individual herds, showing the percentage of infection, and second, it will give us an opportunity to contact individual owners and advise control methods which will be a big step towards the control of this disease in our state.

If, in the future, we find it advisable to pass specific regulations in regard to the control of this disease, it will be done, but we feel that the above plan is a practical way to attack the disease at the present time.

This plan has been in operation only a short time. All records of these tests are filed in the state office, so that a record is kept on each herd. It is too soon for me to draw any definite conclusions or to make a report that would mean anything on the results obtained from such a plan, but I can say that the practitioner has an important part to play in this plan, and I think in any plan that has been devised, and it is up to the practitioner to help control Bang abortion disease.

Discussion

Dr. T. I. Means: Have you done any work on range herds?

Dr. W. H. Hendricks: We are doing a little work on the range herds. In our state we have semi-range herds; in some cases they are out on the range in summer and are brought in in the fall and winter and mingle with the dairy herds. We have found range herds with quite a high percentage of infection. At the present time we have done very little work on range herds outside of one or two herds that are under supervision.

Dr. W. T. Spencer: I was not in the room at the start of this paper; I was down in another section, but I am interested to know if you have had any experience in handling herds through isolation only, and what success has been attained.

Dr. Hendricks: We are just getting started on this plan. I made the statement that we have little legislative appropriation. We are attempting to start a plan on the practical basis of sanitation and education. Our idea is to educate the dairymen as we go. We are just starting in a small way by establishing a demonstration herd here and there throughout the State, getting a few clean herds, and then getting their neighbors to see the value of maintaining a clean herd. Gradually we expect that spirit to grow from one to another until we can get the whole-hearted cooperation of all of them.

At the present time it is rather hard work to get everyone interested. We are just getting started on the plan and really haven't had much data collected from which to draw any definite conclusions.

DR. SPENCER: We all recognize that abortion is one of our big problems, and every state has it, I guess. Nebraska is, in a way, similar to Oregon and some of these other western states, in a large portion of the stock-raising section. We have had, like the rest of you, experience with contagious abortion in our range herds.

I would like to know if any of the men here from this western section have experience sufficient to justify the statement that contagious abortion can be controlled through isolation of infected cattle? Maybe Dr. Ferguson may be able to give us some light on it. I know he has had a lot of experience in a dairy state, but as to a range section—I am wondering just what his experience has been in the handling of range herds.

DR. T. H. FERGUSON: It has been my experience—and for a long time I have thought that Bang disease, or what we call contagious abortion, and allied troubles, can be better handled by sanitation than any other known method. Just practicing partial segregation and sanitation will be of great help.

For instance, for a number of years I practiced about this plan: When I had charge of the health of some herd we would—in those herds the item of expense wasn't considered as much as it is in the ordinary herd—be permitted to do about as we wanted to. Of course, our job was to get that herd in good health and establish breeding efficiency, and prevent all trouble, as far as possible. We found that by being careful and immediately segregating cows with discharges of any kind, providing separate stalls, whenever possible, or maternity stalls, for cows that has started to make up—maybe the uterine seal would just begin to come away, as it usually does a week or two, or three, before parturition—just as soon as we would see evidence of the uterine seal coming away, we would have the cow segregated and a close tab kept on her.

If a heifer, for instance, is starting to make up before time, as they often do, as soon as the udder begins to swell and the vagina indicates that there is going to be an accident, we don't wait for the accident to occur, but get that animal by herself—segregate her. If she does not have an accident, that is all right, but in most cases a careful herdsman who is intent on getting his herd in a better condition will give good cooperation and, as a rule, a good herdsman has good powers of observation. He can tell when a cow is going to do something extraordinary. If there is a storm of abortion, or trouble in the herd, he is on the watch for cows that might possibly abort. Probably you have all had this experience when you were out to treat a case in the country: The owner or herdsman will call your attention to a cow, and say something like this: "Doctor, I think that cow is going to abort; she acts like she isn't going to carry her calf." That is an indication to get that cow by herself. If you observe closely just those principles alone, you can do a whole lot in controlling this disease.

Frequent blood-testing in connection with this plan will get still better results. I believe that there is no question but that any man who is careful enough and will take pains enough, under any of the several plans along this line, can get good results in handling this disease.

Dr. Larson has demonstrated, beyond any doubt whatever, in Wisconsin, that this can be done on quite an extensive scale. He is creating intense interest among breeders and dairymen in that state. I believe it is a good plan to endorse. Of course, as Dr. Hendricks stated, this plan is in its infancy, but it is the best we know of now in handling this disease, that of sanitation and segregation, or some definite plan of handling the herd in connection with blood-testing.

Now, as to handling range herds. I can see a lot of handicaps there unless they can be handled in this way: Keep the negative cows away from the positive ones in a pasture where they can be looked over monthly or more often.

In Wisconsin we have always had trouble with pasture abortions. Several neighbors will turn out their heifers in one pasture. And in almost every instance, one or more have trouble with the stock in such pastures. Some of

these heifers are infected, and some of them abort while they are in the pasture and they infect the others. When they are taken home, at intervals, they will abort. That is a common occurrence in our state.

It looks to me—I don't know much about range conditions—that if a clean negative herd is kept by itself, there should be no reason why that herd would not stay clean on the range.

To get the very best results with this plan one has to give it study and respect all of the simple parts of the procedure. There is nothing complicated about it; it is just the same method of exercising sanitation.

DR. A. M. MCCAPES: Our work in Oregon has been carried on in a cooperative plan for a good many years now. The abortion control work began in our state along about 1919, when the Veterinary Department and the Dairy Department of the College cooperated in eradicating the disease from the college herd. At that time we put into operation a plan of testing and segregating the reactors and non-reactors in opposite wings of the same barn, and in conjunction with this a plan of sanitation, whereby all cows calved in maternity stalls. These stalls were thoroughly cleaned after each calving.

During a course of two years this plan was partially successful, although a few reactors were found among the clean animals each year. After this plan was found to be only partially satisfactory, the reactor herd and the non-reactor herd were separated in two barns a mile apart, with separate attendants, separate pastures, and everything. This plan worked perfectly—absolute isolation, coupled with good sanitation. The two must be in conjunction to get any definite results, the ultimate goal being the elimination of all reactor animals from the herd and the maintenance of a negative herd only. According to our experience in the state of Oregon, based upon this experimental project, we outlined a plan for field work on contagious abortion throughout the State, and we have put this plan into operation in conjunction with the veterinary practitioners, and some field work which we have conducted ourselves.

Prior to inviting the cooperation of the veterinarians throughout the State, we attempted the operation of this plan on about fifty farms. I am making that an approximate number because I am not sure of the actual number. This plan has worked very successfully as a sanitary plan, coupled with an isolation program. But, as Dr. Ferguson said, it takes intensive attention to details in every respect.

After we had been in the field some two years, the State Live Stock Sanitary Board promulgated a plan and regulation whereby herds could be accredited as abortion-free, the same as we did with tuberculosis, by testing and elimination of reactor animals. We have tested some sixty-five thousand blood samples this year, and the percentage of reactors is markedly decreasing in herds in which work has been carried on. The percentage of reactors in the state of Oregon is approximately the same, according to our testing, because new herds are continually being tested; but in herds which have been tested previously, the percentage of reactors is decreasing rapidly and many of those herds are clean upon retests two or three months after the original test is made. In other words, a test today and a test thirty days later, with a recheck seventy to ninety days later, often reveals a clean herd, providing you are not having a storm of abortions.

If you begin testing the cows in a herd in which there are active abortions taking place, or an active spread taking place, you cannot control abortion disease with anywhere near the success that is possible with a herd that is quiescent. We all know that this condition holds: That abortion disease is active at one time and quiescent at another. It is much more simple and probably decidedly more simple, to eradicate abortion disease in a quiescent herd.

That is the plan and the program which we are trying to put over—testing, but testing, if possible, at a time when the disease is quiescent. That, of course, is a very difficult thing to decide definitely until you have a complete history of the herd and have made a couple of tests. But if you are testing at a time when the disease is active, it is more difficult to control, although entirely possible.

DR. M. E. SPRATLIN: Recently we have done a great deal of abortion testing, and to our surprise, in several herds in which we had no clinical evidence of having had abortion disease for a period of four to five years, we found a very, very high percentage—in one case it ran seventy-one per cent, as I recall it—in which there had not been a single physical evidence of contagious abortion. By that I mean that they had not had any problem of sterility. They had had some calves. But there was no question about it, we checked and double checked, and those cows that were positive remained positive. Can any of you account for that?

DR. MCCAPES: I have found that condition in several of the herds that we have been working on, a high percentage of reactors with no particular sign of trouble from infection. That is what I would call a quiescent herd. The disease is there, probably without question, but it is not virulent enough at that time to cause serious trouble. It is not uncommon to find quiescent herds in which a high percentage of reactors will be found. I don't know whether it is the resistance of individual animals or conditions other than that.

DR. SPRATLIN: That was the only thing that was probably abnormal in this particular herd. This herd of cows, which numbered approximately 175 head, had been developed from an original foundation stock, of ten cows, probably twenty years ago. These people had never purchased any outside cows; they had purchased bulls, but never any outside cows. They had sold considerable, of course.

That condition doesn't exist in many herds in the country. It was our conclusion that possibly they had developed a very high natural immunity by having been bred up from an original foundation.

DR. MCCAPES: Was that herd ever tested before?

DR. SPRATLIN: Not until a year ago.

DR. T. I. MEANS: May I ask, how long do positive reactors continue to react, as a rule? Always? Or do they show negative after so many times of testing?

DR. MCCAPES: I knew somebody would ask that question! (Laughter)

To my knowledge, and to all practical purposes, a reactor animal is always a reactor. We have had experience both ways.

Eleven years ago, when the college herd was highly infected with abortion, and before any regulations of sale of reactors was started, two animals were sold out of that herd to a farmer, a dairyman, a short distance out of Corvallis. Last spring those animals were tested for the first time since they were sold, and they were abortion reactors. They were known to be reactors from an infected herd when sold; and they are still reacting, eleven years later.

I have worked with only one herd in the state of Oregon that I know definitely, and have checked definitely on, in which reactor animals have ceased to react in any short period of time. These animals failed to react three months later, but after a second exposure became reactors again and aborted. These animals are still reactors, but they did come down to a negative at one time and then pick up the infection again.

It is, I think, more or less common knowledge that some low-titre reactors will become negative, also that some low-titre reactors will become higher reactors, will react in higher dilutions. The low-titre reactors are what we call "suspicious," and will fluctuate both ways in about fifty per cent of the cases. But a good definite reactor, a reactor in all three dilutions is, to all practical purposes, usually a reactor for life.

However, once in a while, as I said, they will come down to negative, but as far as any practical purpose is concerned, they will not do so.

DR. MEANS: Do you consider all positive reactors spreaders?

DR. MCCAPES: No.

DR. MEANS: Is there any way of determining which reactors are spreaders and which are not?

DR. MCCAPES: Sometimes in a laboratory you can pick out spreaders, by animal inoculation and by isolation of cultures. But I am sure that not all animals are spreaders. However, all reactors are potential spreaders, at calving and aborting time, they then are giving off uterine discharges.

DR. MEANS: Is it advisable, if you are trying to clean up a herd, to get rid of all positive reactors?

DR. MCCAPES: Yes. Dr. Hendricks has explained that pretty well in his plan.

DR. MEANS: Wouldn't you consider some of the positive reactors more valuable cows? That is, if they went through an apparently acute stage of abortion and have gotten over it, than some new cows that you might go out and pick up?

DR. MCCAPES: In an infected herd, yes; in a herd in which you are trying to eradicate that, no. It makes a difference on what plan you are working. If you are working with a mixed herd, your reactors that have recovered are the most valuable, but that isn't the point—we are trying to eliminate the disease in our work, and the keeping of reactor animals is a potential menace in every case.

I believe that we are overlooking one thing in our discussion here—we are talking about reactors only.

Reactor animals, over a period of time, regardless of how valuable they are, are a loss, because the production from those animals will be so decreased in milk, and in calves, that they will not be profitable. That may not be true in one year, it may not be true in two years, but over a period of time they will become so unprofitable that it is not economical to keep them.

The milk production from the herds in which we have been working has averaged about twenty-five per cent less in infected herds than in herds free from abortion disease. Twenty-five per cent of the production of a herd is all the profits. If you take it from that angle it is not economical to keep reactor animals any longer than is necessary. An abortion-free herd is a much more economical production unit.

A. V. M. A. AUTOMOBILE EMBLEM
(Slightly reduced in size)

SALMON POISONING*

By B. T. Simms, C. R. Donham, J. N. Shaw *and* A. M. McCapes

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Introduction

Since the Pacific Northwest was first settled by whites, the question of whether salmon or trout would cause a highly fatal disease of dogs has been very much discussed. The practical dog-owners living along the streams of northwestern California, western Oregon and southwestern Washington, have always maintained that salmon poisoning was a definite and distinct disease. Theorists have claimed that no such disease existed and have pointed out the fact that the same genera of salmon and trout, which are found in this region, form a staple part of the diet of dogs in Alaska and some sections of British Columbia. Suckley,[1] in 1855, suggested that salmon poisoning might be a form of dog distemper. Pernot,[2] in 1911, studied the disease and reported finding an ameba which caused it. Wyatt,[3] in 1925, reported experiments in which he claimed that the disease was produced through use of an ether extract from salmon muscle. He reported having produced an immunity against the disease through subcutaneous injection of such an extract.

It was not until Donham[4,5] reported finding an intestinal fluke in dogs dying from this trouble that any definite knowledge as to the real cause of the disease was available. He found an encysted form of this fluke in both salmon and trout, produced the disease through feeding such material, and recovered mature parasites from his experimentally fed animals. Chapin[6,7] described the fluke, naming it *Nanophyetus salmincola,* Chapin. Ward and Mueller[8] found the encysted form of the parasite in trout and suggested the name *Distomulum oregonensis.*

Distribution

The geographical distribution of the disease includes nearly all of the state of Oregon west of the Cascade Mountains, the southwestern portion of Washington extending about as far north as Olympia, and the northwestern part of California. Veterinarians and dog-owners living in areas immediately adjacent to these

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have almost uniformly reported the absence of the disease in their districts.

Hosts of the Parasite

Mammals: The parasite occurs in at least one species of each of the five families of terrestrial carnivores which are indigenous to the Pacific Northwest. Dogs, Pacific raccoons (*Procyon lotor pacifera*) and mink (*Mustela vison energumends*) have been found which were infested naturally. Cram[9] collected specimens from the coyote (*Canis lestes*), the Pacific raccoon, and the bob cat (*Lynx fasciatus*). Bob cats (*Lynx fasciatus pallescens*), house cats, Pacific raccoons, eastern raccoons (*Procyon lotor*), coyotes,

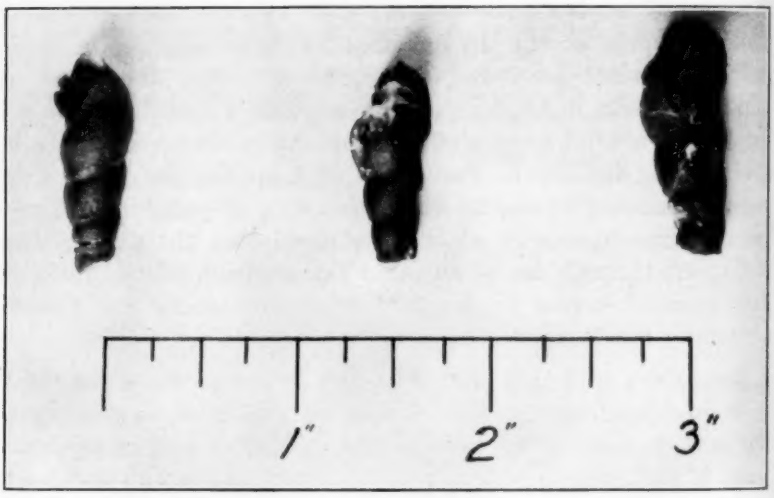

Fig. 1 *Goniobasis plicifera* var. *silcula*, Gould, the molluscan host of the salmon poisoning fluke.

silver black fox (*Vulpes fulvus*), blue fox (*Vulpes lagopus*), guinea pigs, one white rat and one bear (*Ursus americanus*) have been infested through feeding infested fish. Attempts at infestation in chickens, ducks, and a single hog have failed.

Snail: Donham, Simms and Shaw[10] showed that the molluscan host of the parasite is *Goniobasis plicifera* var. *silicula*, Gould. (See figure 1.) This discovery explained the geographical distribution of the disease, as the snail in question has been collected only in those sections where salmon poisoning has occurred. Dall[11] reported this mollusk from British Columbia but as far as is known, specimens have not been collected from that province.

Fish: The fish hosts include all the genera and species of the Salmonidae family which occur in streams of western Oregon. These are cutthroat (*Salmo clarkii*), rainbow (*S. iridiens*), steelhead (*S. gairdnerii*), and eastern brook (*Salvelinus fontinalis*) trout and chinook (*Onchorynchus tschawytscha*), silverside (*O. kisutch*), and chum or dog (*O. keta*) salmon. Examinations of

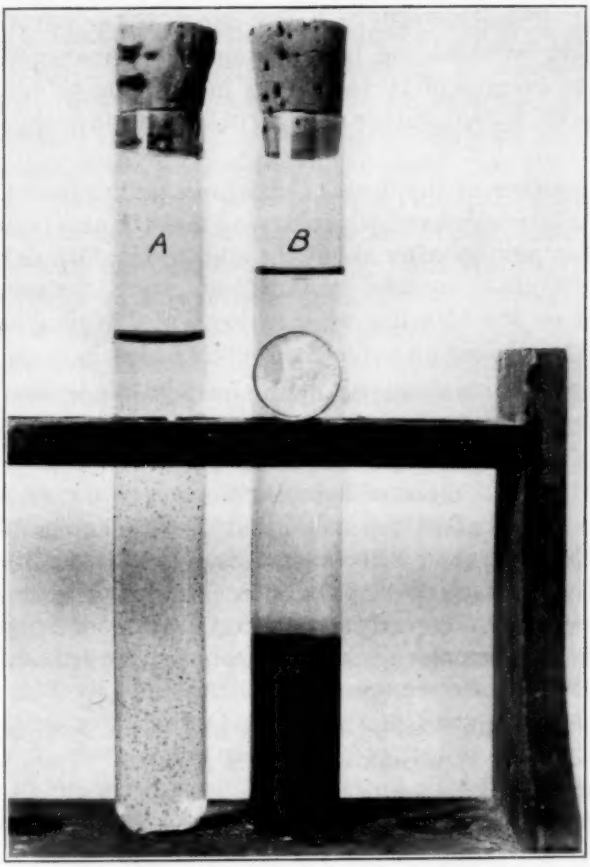

FIG. 2 (A) Specimens of *Nanophyetus salmincola* Chapin suspended in water. (B) Approximately 200,000 flukes obtained from single experimentally-fed dog at autopsy. (Circle shown in tube B is the size of a copper cent.)

other species of fish caught from the same streams have been uniformly negative.

THE PARASITE

This distomatous fluke is from 500 to 700 microns long and of a white or yellowish-white color. It is seen with difficulty with the unaided eye in the intestinal content but is rather easily ob-

served if such material is suspended in water in a glass container. (See figure 2.) It develops very rapidly. Eggs appear in the feces of dogs fed experimentally from the fifth to the eighth day following feeding. Animals destroyed at this time have contained parasites which were almost mature in size but having only one to three eggs in each individual. Such parasites moved rather vigorously when placed in warm physiological saline solution. Experimentally infested dogs, destroyed about the fifteenth day after feeding, have contained fully mature parasites, having an average of 10 to 15 eggs in each individual. These flukes have moved rather slowly in warm physiological salt solution.

Examinations of the feces of experimentally infested animals at regular intervals have demonstrated that the eggs in such feces diminish in number after about the 50th to the 75th day. They usually disappear entirely by the 250th day. A single animal examined on the 95th day after experimental feeding contained parasites which had an average of only 2.8 eggs in each.

These eggs are oval-shaped, approximately 75 microns long and 50 wide. They are a yellowish-brown color with a very indistinctly marked operculum.

Price,[12] in 1928, reported hatching these eggs in approximately three months. Later[13] he advised that the eggs hatched from the 66th to the 139th day. Observations have confirmed his findings. In one instance miracidia appeared as early as the 58th day after eggs were collected, washed and stored at room temperature. Attempts at hatching eggs kept at room temperature during the summer months have uniformly failed.

These miracidia possess long cilia and swim very rapidly as compared to the miracidia of *Fasciola hepatica*. They have not been observed in the act of attacking the snail host.

Sinitsin,[14] of the Zoological Division, Bureau of Animal Industry, U. S. Department of Agriculture, definitely identified the parthenogenetic forms of this fluke. The authors[15] had previously observed four rather frequently occurring cercariae in the molluscan host but had not determined which of these was the salmon-poisoning parasite. Sinitsin further demonstrated that the cercariae attack any portion of the fish and enter by penetrating any of the exposed surfaces.

The number of such cercariae which a single snail may contain is almost unbelieveable. While no counts have been made it is

conservatively estimated that at least 10,000 have been observed in a large snail.

Experimental infestations of snails have not been attempted. Examinations of several hundred snail specimens made during every season of the year have shown quite definitely that those snails under .7 inch in length for the first three whorls do not usually contain any mature cercariae, while such forms are fairly common in the larger specimens obtained at the same places and time and examined in the same manner. It seems probable, therefore, either that the miracidia do not attack small snails or that a considerable length of time is necessary for the complete development of the parthenitae in the snail.

The encysted cercariae (metacercariae) are found in practically all parts of the fish. Donham, Simms and Miller[16] observed them in the kidneys, muscles, gills and livers; and Ward and Mueller[8] observed them in the eyes, the optic nerves, the brains and the walls of the digestive tubes as well as in the kidneys, muscles, gills and livers. Examined alive these cysts are approximately 205 microns in diameter with variations from about 170 to about 255 microns. The smallest cysts contain parasites with unfilled bladders while the larger specimens have very large and well-filled bladders. This observation indicates that the smaller ones are young forms. This conclusion has been substantiated through experimentally exposing fish. Thus a rainbow trout about 30 mm. long, exposed October 4, 1929, and examined four days later, contained 26 parasites, 20 of which were smaller than the average size and had unfilled bladders.

The length of time that the metacercariae live in fish has not been determined. It has been repeatedly demonstrated that the majority of the salmon and trout hatched in the fresh water streams of western Oregon are heavily parasitized before going into salt water. Examinations of ocean-caught salmon have been uniformly negative for parasites. Blueback trout (*Salmo clarkii*), caught soon after returning from the salt water into fresh water, have been only very slightly parasitized. Other trout caught from the same streams have contained large numbers of cercariae. These observations indicate that the encysted parasites do not live indefinitely in the fish host.

The distribution of the cysts in the fish hosts depends somewhat upon the age and size of the fish. In younger fish less than 2 inches in length it is unusual to find any considerable number of cysts in the kidneys, while in the larger specimens parasites are

more numerous in the kidneys than in any other portion of the body. There is a definite tendency on the part of the metacercariae toward arrangements in chains somewhat similar to the short chains of streptococci. Whether this is due to a tendency on the part of the parasites to follow the tunnels already made by other cercariae has not been determined.

These cysts have lived for 50 to 75 days when stored in fish at temperatures slightly above freezing. Viable metacercariae have been demonstrated through feeding such stored fish when no movements could be observed in the cysts by placing them on a warm microscopic slide. Donham, Simms and Miller[16] destroyed the parasites by exposure to below-freezing temperatures.

In attempts to preserve metacercariae for experimental feeding, kidneys of chinook salmon were collected at an egg-taking station, September 28, 1929, and stored at about 37° F. for 12 days. By this time the kidney tissue had disintegrated considerably and was very soft. It was chopped up with scissors and washed through a wire screen with 40 meshes to the inch. The encysted flukes were slightly heavier than the kidney tissue and settled to the bottom rather rapidly in physiological salt solution. After several washings the parasites were relatively free from extraneous material. A part of these were suspended in .85% sodium chlorid solution and another part was placed in tap water. Both were stored at approximately 37° in stoppered vials. In both groups the liquid in which they were suspended was changed approximately once a week. At the end of five and one-half months, motion could still be observed in a few cysts in each vial. At the termination of six months no moving parasites could be observed in either group of cysts.

Attempts at liberating these encysted parasites through digestion with artificial gastric juice were negative, but artificial pancreatic juice digested the capsules and liberated the flukes.

Pathogenicity

Attempts have not been made to determine experimentally whether these parasites are pathogenic for the molluscan host. The fact that the largest snail specimens are the ones most frequently parasitized and the further fact that snails are extremely numerous in streams in which infestation is very common, indicates that they tolerate this parasite very well.

It seems reasonable to expect at least some damage to the fish host when large numbers of these parasites are present. Donham,

Simms and Miller[17] suggested such a possibility but did not produce any evidence to support the suggestion. Ward and Mueller[8] found eastern brook trout which were dying in large numbers in a hatchery were heavily infested with these parasites. Examination showed that the dead and dying fish were more heavily infested than were the apparently normal ones in the same ponds. They drew the conclusion that these parasites were causing the losses among these fish.

Observations made by the writers at this same hatchery have shown that some fish have died when the numbers of parasites contained were not very large. Apparently healthy fish obtained from other sources have contained just as many encysted flukes. More evidence is necessary before any definite statements can be made concerning the pathogenicity of these parasites for the fish host. On the other hand, it has been quite definitely established that fish can tolerate large numbers of these cysts. Counts of more than 200 parasites per fish have been made quite often in apparently healthy rainbow trout less than two months old and measuring less than 1¾ inches in length. In a cutthroat trout about 14 inches in length, more than 2500 parasites were counted in 65 mg. of kidney tissue. In another cutthroat 4 inches long, 174 parasites were counted in the kidney tissue. A complete count of still another cutthroat 4.6 inches in length revealed 14,062 parasites. All of these fish were apparently healthy and the last three mentioned were caught on a hook.

Pathogenicity for the mammalian host seems restricted to the members of the Canidae family only. Donham, Simms and Miller[16] reported the dog, the silver black fox, and the blue fox as being susceptible, and Donham and Simms[18] observed the disease in experimentally fed coyotes. No other species of Canidae than these four have been studied.

Two bob cats exhibited a slight loss of appetite on the sixth day after experimental feeding. This lasted for three days. Neither animal was visibly sick, however. The one bear, a cub estimated to be three months old, which was fed, lost weight after infestation. It showed no other effects and gradually returned to good physical condition. In all the other experimentally fed animals not belonging to the Canidae family no symptoms whatever have been observed.

The Disease

Causal agent: While it has been definitely established through the experimental feeding of more than 100 dogs that the disease

is associated with infestation with the fluke in question, the exact causal agent has not been determined. The acuteness of the attack, the severity of the lesions, and the definite immunity following a recovery are certainly suggestive of an infectious agent of some type. Donham[19] attempted to isolate pathogenic bacteria from the various organs of both sick and dead animals, with negative results. Blood smears have not revealed protozoan parasites.

The introduction through a stomach-tube of the intestinal contents of a dog dead from the disease into the stomach of a susceptible animal did not produce the trouble. Kennel exposure of susceptible dogs to those suffering from salmon poisoning has uniformly failed to produce salmon poisoning.

Two dogs were injected intraperitoneally with blood from dogs which were showing severe acute symptoms of the disease. In the first animal so injected no symptoms whatever developed. The animal later proved susceptible when fed infested fish. The second animal so injected developed symptoms similar to salmon poisoning and died. Lesions were somewhat comparable to those seen in dogs dead of the disease, with the exception of their being much less severe.

The intraperitoneal injection of flukes obtained from the intestines of dogs dead of the disease resulted in the development of symptoms somewhat comparable to those of salmon poisoning in 17 out of 19 susceptible dogs receiving such injections. One of the two which did not develop symptoms received only 100 flukes. Of the 17 which developed symptoms, 14 died. Of these, 3 were not autopsied. Nine of the 11 examined postmortem had lesions indicative of salmon poisoning. The other two had abscesses and pyemia.

Two immune dogs which received such intraperitoneal injections did not develop any symptoms. Flukes which were heated to 60° C. for an hour, those boiled for three minutes, and those frozen for 7, 24 and 39 days, respectively, failed to produce any disturbance when injected intraperitoneally.

A susceptible dog, which received an intraperitoneal injection of rediae and cercariae obtained from snails, showed no disturbance following the injection.

Incubation period: Susceptible dogs develop symptoms of the disease from six to nine or ten days after eating parasitized fish. Data on dogs fed experimentally indicate a little longer incubation period during warm weather than during cold.

Symptoms: The onset of the disease is very sudden. Animals which have been apparently normal will show a complete loss of appetite as the first noticeable change. Temperatures taken at this early stage usually are above 103.5° F., ranging up to as high as 106. By the second day the affected animal is usually quite inactive. It shows a tendency either to sit on the haunches or lie down. In most instances the temperature remains high until about the third day after symptoms have occurred. Beginning at about this time there is a gradual drop, with a tendency toward reaching normal about the sixth to eighth day after the disease appears. About the third to fifth day, the eyes usually show more or less of a purulent discharge. There may be some edema of the lids, which causes the eyes to have a slightly sunken appearance.

After the third or fourth day, vomiting may be a very prominent symptom. Those animals which show this disturbance usually vomit immediately after drinking water. In such cases the thirst is very marked and the animals will drink large quantities of water at very frequent intervals. Apparently almost none of it is retained.

The bowels are usually somewhat sluggish for the first four or five days. At the fifth or sixth day, diarrhea may appear. The stools are at first soft and mixed with mucinous material. About 24 to 48 hours after diarrhea sets in, blood usually appears in the feces. The amount varies from just enough to make a distinct discoloration to almost pure blood.

At about the sixth or seventh day, or when the temperature has reached approximately normal, the affected animals frequently appear very much brighter. Sometimes they take food and show every indication of marked improvement. In most instances, however, this is of very brief duration. The temperature continues to drop, the animals continue losing strength, and in another 24 to 48 hours death occurs.

Mortality: The mortality in experimental cases has been well above 90 per cent. Dog-owners in the districts where the disease is prevalent have reported anywhere from as low as 50 to 60 per cent up to at least 90.

Lesions: The lesions of the disease are confined to the digestive tube. The entire intestine usually shows more or less inflammation. The ileo-colic valve, the rectum, and the lower portion of the ileum are frequently the most seriously affected, but in some cases the upper portion of the small intestine exhibits the most

severe lesions. The inflammatory reaction may be very severe, with a large amount of hemorrhage into the lumen of the bowel. Flukes are usually present in the bowel content in considerable numbers. This naturally varies, however, with the severity of the infestation. In experimental work as many as 200,000 specimens have been recovered from a single animal. (See figure 2.) At the other extreme, dogs have died after receiving 100 parasites. In such cases it was very difficult to find any flukes in the bowel content.

In most cases the ileo-cecal lymph-gland is considerably enlarged. Occasionally some of the mesenteric lymph-glands are larger than normal.

Microscopic sections reveal the presence of flukes buried in the mucosa of the duodenum but not in other portions of the intestine. Such parasites have not been found beneath the muscularis mucosa. Necrosis of the superficial portions of the mucous membrane is usually evident in sections from all portions of the bowel. Hoeppli[20] attributed the necrosis to the sucking and boring action of the parasites.

Diagnosis: Perhaps the most significant symptoms in regard to diagnosis are the very sudden onset of the disease with complete and persistent loss of appetite and the vomiting and diarrhea in the later stages. A definite diagnosis can usually be established through a microscopic examination of the feces. Eggs appear just about the time the first symptoms are noted and continue to be present in the excrement until the termination of the disease.

Treatment: Treated dogs have consisted of two groups, namely, those fed infested fish and those which had eaten fish accidentally and were presented for treatment after symptoms had developed.

In the experimentally infested animals, attempts have been made to destroy the flukes, both before and after symptoms of the disease appear. In some instances medicinal treatment was administered within twenty-four hours of the time that the parasitized fish was eaten. Among the medicinal agents which have been tested are aspidium, areca nut, calomel, carbon tetrachlorid, tetrachlorethylene, magnesium sulfate and castor oil. While an occasional treated dog has recovered, it cannot be said that any of the medicinal agents given have been successful.

Two dogs were given serum from an immune animal. In one instance, this was injected before symptoms developed and in the other case after symptoms had appeared. Both dogs died and showed typical lesions of salmon poisoning on autopsy.

Immunity: It is well established that dogs which recover from an attack of the disease are immune thereafter. At least some flukes develop and produce eggs in such immune animals if they consume parasitized fish. While some dog-owners think an occasional animal is immune from the time of birth, all kennel-raised dogs used in the experimental work have been susceptible. These have included some thirty animals, at least ten of which were from an immune dam.

Fourteen dogs were used in attempts to produce immunity through repeatedly feeding small numbers of parasites. Even after feeding sublethal numbers over extended periods of time, all these animals developed the disease and died after they were exposed to heavy infestation.

The three dogs mentioned above which developed symptoms and recovered following intraperitoneal injections of mature flukes were fed fish containing large numbers of living parasites. One is still alive ninety days after being fed. It has passed large numbers of eggs for the past eighty-odd days. The second dog of this group developed no symptoms until about the twenty-eighth day after the fish was consumed. At that time it showed indications of fright disease and died on the thirty-second day with typical symptoms of this trouble. Autopsy did not reveal any lesions of salmon poisoning. The third dog remained apparently normal for fourteen days after consuming the fish. Large numbers of eggs were present in the feces after the seventh day. Temperatures were taken daily and were normal. The appetite was good and the animal was playful. On the morning of the fifteenth day, the dog was found dead. Autopsy revealed a full stomach, a condition almost never found in salmon poisoning. There were no lesions of this disease. While two out of these three dogs died following exposure to the parasites, it seems probable that all three animals were immune against salmon poisoning.

The injection of flukes boiled for three minutes, flukes heated to 60° C. for an hour, and flukes frozen for from seven to twenty-seven days not only failed to produce symptoms as previously mentioned but also failed to protect when the dogs were later fed parasitized fish.

Donham[19] failed to produce immunity through the injection of serum from an immune dog. Wyatt[3] reported the establishment of immunity through the subcutaneous injection of an ex-

tract from salmon muscle. Simms, Donham and Shaw[21] failed to protect a dog with this material.

Prevention: The only satisfactory means of prevention known at present consists in handling dogs and foxes in such a manner that they will not have an opportunity to consume any salmon or trout originating in fresh water streams of the infested districts. Dr. F. C. Myers, of Corvallis, Oregon, and Dr. J. L. Masson, of Eureka, California, both practicing veterinarians, have reported apparent success following the use of apomorphin within a few hours after dogs had eaten parasitized fish. One experiment dog, treated according to their directions within three hours after the parasitized fish was eaten, did not develop symptoms of the disease. It later proved to be susceptible when given a second feeding of parasites.

Discussion

While the disease is mainly of local interest in the Pacific Northwest, it should be borne in mind that with present means of transportation, practicing veterinarians in any section of the country may possibly be called on to treat a case of salmon poisoning. It would be a very easy thing for a tourist to have his dog consume parasitized fish in Oregon and then present the animal some ten days later to a veterinarian on the Atlantic seaboard.

Both fish and mammalian hosts for the fluke are fairly widely distributed throughout the world. Apparently, then, all that is necessary for the disease to become widespread is the establishment of snail hosts in other areas. There are two possible methods of such an occurrence. One is through a gradual spread of the snail host which has already been described. Since there has never been a very complete survey made for distribution of this snail, it cannot be said whether it is invading new territory. It seems probable, however, that it is not progressing in a northerly direction as the first specimens collected and described by Gould, nearly a hundred years ago, originated at Fort Nisqually, Washington. The snail is not found today very far north of this point.

The second possibility is the establishment of other species of snails as hosts. It is well known that many of our flukes can develop in more than one molluscan species so that it is not beyond the realms of possibility that there are suitable snail hosts in other sections which are only waiting for miracidia of *Nano-*

phyetus salmincola. If such hosts exist on the North American continent, it is quite probable that the increased traffic in dogs and fur-bearing carnivores will sooner or later expose such hosts in areas which are at present free from the parasite.

Salmon poisoning has evidently been a limiting factor in the distribution of species of Canidae in the Pacific Northwest. Suckley[1] reported coyotes as being very prevalent in eastern Oregon. He stated they fed upon spawned salmon which were along the streams. He did not mention these animals as occurring in western Oregon. He mentioned the gray wolf specifically as not being present in western Oregon except on the Clatsop plains and in one section of the Cascade Mountains. He further recorded finding foxes in eastern Oregon without mentioning any specimens collected in the western part of the State.

His early observations agree with statements which have been had from many of the older settlers of western Oregon. They have advised that coyotes were almost unknown in the Coast Mountains and along the coastal streams of the State until after the commercial canning industry was well established and the number of salmon which spawned and died in the streams had decreased very materially.

Further studies of the effects of the parasites upon the fish host seem very desirable. Even though it may be established that they do not cause any considerable death losses, it is quite possible that moderate or severe infestations may result in retarding the normal growth of both salmon and trout. If this does happen, the parasite is of very considerable practical importance both in the commercial fish industry and in game-fish propagation.

Further studies must be made before the definite cause of the symptoms and lesions can be determined. Hoeppli[20] thought the sucking and boring action of the flukes resulted in the lesions observed in the digestive tract. Since the parasites are found buried in the mucosa of the duodenum only, this would not explain the lesions in all other portions of the intestines.

Since a very definite immunity follows recovery from an attack of the disease, it seems that means of producing this in a practical manner should finally be worked out. If this can be done, it will be of much importance to the live stock industry of the regions affected, as the dog is a necessary part of the equipment of the ranches in these sections.

SUMMARY

1. Salmon poisoning is a disease of Canidae associated with infestation with the trigenetic fluke, *Nanophyetus salmincola*, Chapin.

2. The mammalian hosts include at least one species of each of the five families of terrestrial carnivores which are indigenous to the Pacific Northwest. The snail host is *Goniobasis plicifera* var. *silicula*, Gould. The fish hosts include all genera and species of Salmonidae which occur in the streams of western Oregon.

3. Mammalian hosts void eggs as early as the first or sixth day and as late as the one hundredth to the two hundred fiftieth day after infestation.

4. The snail host can tolerate large numbers of the parthenitae. Apparently healthy salmon and trout may contain very large numbers of metacercariae. Among the mammalian hosts the typical syndrome of salmon poisoning has been observed in Canidae only.

5. The causal agent of the disease is unknown.

6. The intraperitoneal injection of mature flukes has resulted in a condition similar to salmon poisoning.

7. No satisfactory medicinal treatment has been found.

8. A definite immunity follows from an attack of the disease.

9. No practicable method of immunization has been found.

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New Bulletin on Laurel Poisoning

Technical Bulletin 219-T on laurel poisoning, just issued by the U. S. Department of Agriculture, reports a study of the poisonous properties of the plants and tells how to treat poisoned animals. Toxic properties of mountain-laurel and sheep laurel have been recognized for nearly 200 years. In each the poisonous principle is andromedotoxin.

Dr. C. Dwight Marsh and A. B. Clawson, physiologists of the Bureau of Animal Industry, fed leaves and flowers of mountain-laurel to sheep. While dried plants were used, the dosages were computed in terms of green plants. These varied from 0.15 to 1.20 per cent of the weight of the animal. Results were not entirely consistent. In some cases doses of 0.60 per cent did not cause sickness, while in one case a dose of 0.35 per cent resulted in symptoms of poisoning. In another case a dose of 0.50 per cent caused death in less than 48 hours.

Tests with cattle and goats showed much the same results as with sheep, the animals first showing depression, then weakness, nausea, and sometimes prostration. Effects of sheep laurel where similar to those of mountain-laurel, as was expected, because of their similar toxic properties, although symptoms developed more quickly from sheep laurel, and the effects were more prolonged. The investigators did not feed the laurels to horses.

Cattle, sheep and goats are susceptible, but as a rule the danger is relatively slight, because animals rarely eat laurel in quantity if other forage is available. Usually the most serious losses occur in spring, before the grass has had time to grow.

Linseed oil has been used as a remedy and with good results. Castor oil and lard, sometimes with milk added, purgatives, emetics, and balls of butter have all proved effective. Grease and oil, however, have been found to be the best remedies.

Technical Bulletin 219-T may be obtained free from the Office of Information, Department of Agriculture, Washington, D. C.

INFECTIOUS LARYNGOTRACHEITIS OF FOWLS*

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The term "infectious bronchitis," as commonly applied to a specific, acute infectious disease of the respiratory organs in fowls, is a misnomer. May and Tittsler[1] first called attention to this disease in 1925, and described it as a tracheo-laryngitis, and rightly so, since the disease does not affect the bronchi alone so frequently as it does the trachea and the superior larynx. Due to the fact that the latter two organs seem to be the main seats of infection, "infectious laryngotracheitis" would be a more suitable term to designate this disease which has come to be generally known as infectious bronchitis. Although May and Tittsler were the first to recognize infectious laryngotracheitis as a distinct entity and described it as such, it had been suggested by Kaupp,[2] in 1917, that such a disease existed among fowls. And as early as 1899, Salmon[3] mentioned a respiratory disorder resulting in a high mortality among fowls, which, from his description of the disease, would lead one to believe that he was dealing at that time with laryngotracheitis as we know it today.

It has been only within recent years that the state of confusion which always existed relative to respiratory diseases in fowls has become somewhat clarified. For years there was considerable controversy regarding the identity of chicken-pox and avian diphtheria, although it has now been proven beyond all question of doubt that the virus of chicken-pox does produce warty-like lesions on the skin of the head as well as canker-like membranes on the mucosa of the mouth. It is altogether possible that a large majority of cases of so-called avian diphtheria were, in reality, infectious laryngotracheitis instead of chicken-pox. So, in all probability, infectious laryngotracheitis is a new disease only from the standpoint of differentiating it from other fowl diseases.

One is justified in arriving at this conclusion after having reviewed the early work of Gwatkin,[4] in 1924, and Beach,[5] in 1925, on this disease. The former at first considered it a very acute type of chicken-pox caused by chicken-pox virus. In his report[6] a year later, however, Gwatkin discussed the disease as a

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tracheitis of unknown origin. Beach,[5] in 1926, not only produced chicken-pox by applying the tracheal exudate of infectious laryngotracheitis to the scarified comb of cockerels, but also produced laryngotracheitis in cockerels by introducing chicken-pox virus into the trachea of the birds, although he arrived at no definite conclusion relative to the etiology of the disease.

Susceptibility

Susceptible animals: The disease affects fowls much more frequently than any other of the feathered species, although lesions resembling those of infectious laryngotracheitis in fowls have been observed upon postmortem examination of turkeys, ducks, pigeons and such free-flying birds as quail, blackbirds and sparrows. No trials have been made, however, to determine the susceptibility of these birds to the disease.

Age of susceptibility: Infectious laryngotracheitis may attack fowls of any age. It has been observed in chicks eight days old and in hens four years old. The period of greatest susceptibility, however, appears to be between four and eighteen months.

Severity of infection: The mortality in flocks affected with laryngotracheitis varies within wide limits. Flocks under the author's observation, in which ninety per cent of the birds were affected, have recovered with a loss of less than five per cent, while in other flocks the mortality has been known to reach seventy-two per cent. The average mortality, however, is about twenty per cent.

Factors favoring infection: The disease occurs at all times of the year in congested poultry districts. Climatic conditions and seasonal changes do not appear to be factors in its development. Extremely severe outbreaks have occurred during warm, dry weather as frequently as in cold, damp weather.

Housing and feeding conditions have little influence upon the spread of the disease, since outbreaks have been observed in flocks that were confined in every conceivable type of house and also fed in various ways.

The condition of the individual fowls has much to do with the incidence and mortality of the disease in a flock. Fowls which are in poor physical condition due to such causes as infestation of external or internal parasites or other complicating factors are more often affected than fowls free from them.

Course of the disease: The disease appears suddenly in a flock, spreads rapidly and, after a duration of ten to fourteen days,

terminates as quickly as it began. The individual fowl either makes a complete recovery or dies in three or four days. The incidence, severity and mortality of the infection varies within extremely wide limits. For instance, in two flocks of the same age, in pens side by side, reared in the same manner, the mortality, for no obvious reason, was 4.6 per cent in one and 27.4 per cent in the other.

Period of incubation: In natural outbreaks of the disease, the incubation period is from seven to twelve days. Artificial infection in young chicks has been produced in less than thirty-six hours.

Symptoms

The most pronounced symptom is the manifestation of extreme respiratory distress shown by the affected fowl, which suddenly elevates the head, extends the neck and opens the mouth in gasping for air. Affected fowls frequently exhibit violent spasms of coughing and sneezing, which sometimes expels the bloody mucus from the trachea. Not infrequently there is a watery discharge from the eyes, which is later replaced by a caseous exudate due to the extensive inflammatory involvement of the eyes and surrounding tissues. The fowls refuse to eat, become listless and develop a temperature frequently in excess of 110° F.

Lesions: Fowls that have died from laryngotracheitis seldom show any pathological changes in any of the internal organs, with the exception of varying degrees of inflammation in the trachea and superior larynx and, occasionally, the lower larynx and bronchi. There is an accumulation of mucus and blood in the lumen of the trachea. Sometimes this is sufficient to obstruct the passage of air, to the lungs, completely, resulting in the immediate death of the fowl from asphyxiation. The bloody mucus undergoes a rapid change into caseous exudate. The forceful expulsion of the bloody mucus or caseous exudate from the upper respiratory tract by sneezing and coughing hastens recovery.

Frequently the eyes and sinuses also are involved. The inflammation of this region adds greatly to the severity of the attack, entailing a high mortality.

Infectivity: That the disease is infectious in nature can be readily demonstrated by introducing into the trachea of a normal healthy fowl a small amount of the tracheal exudate from a fowl affected with laryngotracheitis. Symptoms usually develop in forty-eight to seventy-two hours. Young chicks placed in con-

tact with infected chicks develop the infection within five or six days. The exudate which contains a considerable amount of blood is more virulent than the caseous exudate. The virulence of the contagium varies to a marked degree.

Bacteriology

Cultures made from the heart-blood, liver, spleen, kidney, bone-marrow and nerve tissue, for the most part, have been sterile. A variety of culture media under both aerobic and anaerobic means have been employed. Cultures made from the tracheal exudates were frequently negative, although a variety of bacteria, including *Pasteurella avicida*-like organisms, diplococci, *Pseudomonas pyocaneus* and other unidentified bacteria were found, none of which produced laryngotracheitis by intratracheal instillation.

Distribution of virus: The infectivity of the tracheal exudate is quite readily established by intratracheal instillation. The disease appears to localize in the trachea, since it has been not possible to demonstrate the presence of the contagium in the heart-blood, liver, spleen, kidney, bone-marrow or nervous tissue by subcutaneous or intraäbdominal injection or by instillation into the crop or trachea of suspensions of any of these tissues.

Avenues of infection: Numerous trials, in which subcutaneous injections were given, failed, in all cases, to produce any symptoms of the disease and the birds showed no ill effects from the injection. Intravenous injections, on the contrary, were usually quite harmful. In some instances, the birds died immediately following the injection, while others lived for some hours but at no time showed symptoms of laryngotracheitis. Cultures made from the organs of these birds were sterile, so it was assumed that the birds died from thrombi caused by particles in the material injected.

Beach,[5] however, was able to produce laryngotracheitis by subcutaneous and intravenous injection of tracheal exudate.

Instillation of tracheal exudate into the crop was not detrimental to the treated fowls. The application of this exudate to the scarified comb produced neither the lesions of chicken-pox nor infectious laryngotracheitis.

Filtrability of Contagium

Beach[7] transmitted the disease by intratracheal, intraperitoneal and intravenous inoculations of sterile Seitz filtrates, and stated that the causative agent is a filtrable virus.

Repeated trials have been made to determine the filtrability of the virus, but all attempts have been negative. In this filtration work, Berkefeld W, Chamberland F and L-3 and Seitz filters were used. The tracheal exudate was weighed, triturated into a uniform, pasty mass, then diluted with sterile physiological saline. The suspension was divided into four equal portions and each portion adjusted to a different pH value as follows: 3.0, 4.5, 6.0 and 8.0. The adjusted suspensions were passed each through a separate filter, but all of the same type. Filtration was made by vacuum, using a negative pressure of 15 inches of mercury for Chamberland and Berkefeld filters and 5 inches of mercury for the Seitz filters.

Four lots of chicks were then given a tracheal instillation each of a different filtrate and a fifth lot an instillation of unfiltered exudate. Approximately 0.5 cc of filtrate was administered to each chick, while only 0.1 cc of exudate was used. Both one-day-old and twelve-week-old chicks were used in these trials. The day-old control chicks developed symptoms in forty-eight hours and all died within five days, but in no case were symptoms of infectious laryngotracheitis produced in birds treated with filtrate. Twelve-week-old birds given filtrates adjusted to the above-mentioned pH values by intravenous, intraäbdominal or intratracheal inoculation failed to develop the disease, although control birds given unfiltered exudate intratracheally died of laryngotracheitis.

Carriers: Spontaneous recovery frequently occurs in both natural and artificial infection. Recovery was considered to have taken place with the cessation of respiratory symptoms and resumption of eating, and is usually very rapid. Birds which have recovered do not harbor the contagium for any great period of time. This varies, however, with the individual birds. The trachea of a bird which had recovered was swabbed with a sterile cotton swab, which was then washed in a minute quantity of sterile saline. Approximately 0.1 cc of this suspension was instilled into the trachea of a young chick and this process was repeated daily, thus inoculating a series of chicks, one every twenty-four hours, until no symptoms were produced in the chicks. This took usually five or six days. In a few instances in which the trachea of the previously-affected bird was swabbed at longer intervals, it was found that the contagium was present for at least twelve to fifteen days.

Immunity

Immunity after recovery: As a general rule, a high percentage of birds which contract laryngotracheitis recover spontaneously. A high degree of resistance is produced during the course of the disease against subsequent infection, for in no case has it been possible to reproduce the disease in any of these birds by intratracheal instillation of highly virulent contagium.

Artificial immunity: Attempts to increase the resistance of birds by parenteral injections of modified tracheal exudate against subsequent inoculation of the contagium were made by (1) instillation into the crop, (2) subcutaneous injection of tracheal exudate treated by chloroform vapor, formalin, or heated at 56° C., and fresh tracheal exudate, (3) intravenous, intraäbdominal and intratracheal injections of filtrates made by passing suspensions of tracheal exudate through Berkefeld W, Chamberland F and L-3 and Seitz filters, and (4) instillation of varying dilutions of tracheal exudate into the trachea.

It was not possible to produce the slightest degree of immunity by any of the methods employed against intratracheal instillation of virulent tracheal exudate, administered ten, twenty, or thirty days after the initial injection.

Conclusions

Laryngotracheitis is an acute infectious disease of fowls.

Pathological changes occur principally in the larynx, trachea, and bronchi. These changes consist of inflammation of the affected parts and an excessive amount of mucus and hemorrhage into the lumen of these organs. The contagium localizes in these respiratory organs and in the contained exudate.

Death apparently results from asphyxiation rather than a generalized infection.

The nature of the causative agent has not been determined although rather exhaustive trials were made to culture or filter the virus.

Infection has been produced only by instillation of the contagium into the respiratory tract.

Injections of the tracheal exudate and modified exudate did not increase the resistance of fowls against subsequent infection.

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Fined for Drug Adulteration

A fine of $500 and costs recently was imposed by Judge James G. Morton, of the United States District Court of Concord, N. H., upon a Lancaster, N. H., drug concern for adulterating drugs. The adulterated and misbranded drugs included calomel tablets, tincture of nux vomica, and strychin sulfate tablets.

The fine and costs were levied by the Court upon the P. J. Noyes Co., of Lancaster, N. H. The company pleaded guilty to the government's charges that this firm had shipped from the state of New Hampshire into the states of New York and Massachusetts certain drugs that were adulterated and misbranded in violation of the Food and Drugs Act. In bringing this action the government alleged a second offense, since this firm previously had been prosecuted and fined for violation of the Act.

"The Food and Drug Administration is obliged under the Food and Drugs Act to exert its utmost efforts to insure reliability and accuracy in the drug supply of the nation so that physicians and others who call for certain drugs in case of urgent necessity may use them with a feeling of security," said W. G. Campbell, chief of the Food and Drug Administration, in commenting upon the case. "If the Food and Drugs Act were not capable of insuring this result, it would be ineffective indeed. It is the duty of the administration to use every method within its power to protect the public from manufacturers and dealers who violate the Act. This course will encourage confidence in the nation's food and drug supply."

Since 1907, when the Act went into effect, the government has instituted more than 17,000 seizures and prosecutions for violations of the law. In addition, officials charged with the enforcement of the law have shown American food and drug manufacturers in many ways how to improve the purity and quality of their products, Mr. Campbell declared. "It is my conviction," he said, "that the government's long-time regulatory campaign has had a very salutary effect upon the drug trade of this country."

SOME LESIONS ASSOCIATED WITH PARALYSIS*

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INTRODUCTION

For the last few years, there has been reported a great increase in the amount of paralysis of the legs in chickens from two to twenty months of age. This condition has reached such importance to the poultry-raisers throughout the country that the Committee on Poultry Diseases of the United States Live Stock Sanitary Association for 1929 "especially urged special fundamental research on fowl paralysis." There has been some work done on this condition but much less than has been done on most of our poultry diseases. A number of investigators claim that it is a specific disease, some saying that it is of a hereditary nature, and others that it is infectious. Some workers are of the opinion that the paralysis is merely a symptom and the typical gross and microscopical lesions found in the so-called fowl paralysis, range paralysis, *neurolymphomatosis gallinarum*, neuritis, etc., as the condition has been variously named in literature, are merely complications of either coccidiosis or intestinal worms. Still others admit that coccidiosis and intestinal worms frequently cause paralysis of the legs, but claim that true fowl paralysis with the gross and microscopical lesions as described is a specific disease.

HISTORY AND LITERATURE

The first report of this condition that we have been able to find in literature is by Marek.[1] In a very limited number of birds, he reports enlargement of the spinal nerve roots and lumbar plexus. Microscopical examination of the sciatic nerves, lumbar plexus, spinal cord, and spinal nerve roots showed infiltration of these tissues with mononuclear cells, and almost complete loss of nerve fiber of the lumbar plexus and sciatic nerves. Kaupp[2] reports round-cell infiltration of the spinal cord, atrophy of fibers and sometimes coagulation necrosis; also round-cell infiltration of liver and kidneys. Macroscopic study showed nearly everything normal. He regards the condition as infectious.

*Abstract of a thesis presented for the degree of Master of Science, Ohio State University, August, 1930, while on leave of absence from the University of Wyoming, at Laramie.

Van der Valle and Winkler-Junius[3] found leucocytic infiltration in swollen dorsal ganglia, spinal cord and lumbar plexus, and consider the condition is due to a filtrable virus. May, Tittsler and Goodner[4] worked upon paralysis and found an infiltration of round cells in some of the nerve tissue. Attempts to transmit the disease by contact, feeding, and injections of material from birds with the condition were unsuccessful. They also report negative results from bacteriological efforts at etiological agents. They state that macroscopic study shows nearly everything normal, but the intestines are frequently inflamed or show a thickened wall.

Doyle[5] found swelling and grayish discoloration of large peripheral nerves, plexuses and spinal ganglia. In some cases most nerve trunks in the body showed well marked lesions. He also frequently found a grayish color of the iris, as well as paralysis of the wings and twisting of the neck. The microscopical findings were mononuclear infiltrations of one or more of the following tissues; nerve trunks, plexuses, ganglia, spinal cord, brain, iris and ciliary body. Myelitis and leptomeningitis were rather common. The infiltrations in the central nervous system were unmistakably perivascular. He found these infiltrations also in birds not showing clinical symptoms. He states that the histopathology strongly suggests that the disease is infectious although feeding and injection experiments did not reproduce the disease. In a later article, Doyle[6] believes the cause is a neurotropic virus. In the same work he states that breeding from an experiment flock, where there were cases of paralysis, resulted in paralysis of the offspring, while controls from flocks free from paralysis did not develop the condition.

Beach,[7] De Blieck[8] and many others claim paralysis is due to intestinal worms. De Blieck claims paralysis in Europe is due to *Davainea proglottina*. Beach and Davis,[9] as well as others, state that paralysis of the legs of pullets is a prominent symptom of the chronic type of coccidiosis. Young[10] fed oöcysts to a chick on three separate occasions, one month apart. The first oöcysts were fed when the chick was 45 days old. The bird developed paralysis after the second feeding and continued in this condition until death. He states that due to the conditions under which the bird was kept, the results support the assumption that fowl paralysis is one of the symptoms of chronic coccidiosis. Beach[11] says he has been criticised for using the term "chronic coccidiosis" because a number of investigators doubt that there is such a

condition, because it had not up to that time been produced experimentally. Tyzzer[12] reports he has produced chronic coccidiosis experimentally with *Eimeria acervulina* and also with *E. maxima*. Nothing is said about paralysis in these birds, however.

One of the most recent and extensive works upon paralysis of chickens is by Pappenheimer, Dunn, and Cone.[13] They made extensive studies upon sixty cases in all stages of the disease. Their gross and microscopical findings were very similar to those found by Doyle. Routine stains and Marchii and Pal-Weigert methods were used in individual cases. They found the peripheral nerves showed the most severe lesions, showing infiltration with small and large mononuclear cells and plasma cells between the nerve fibers. In places they were scattering and in other places dense enough almost to replace the nerve tissue completely. The predominating type of infiltrating cell they found to be morphologically identical to the lymphocyte. In some cases the fibers disappeared, their place being taken by a vacuolar or foamy tissue, often filled with coagulated edema and containing free fat globules. They report also that the myelin sheaths appeared collapsed and Pal-Weigert preparations gave evidence of advanced degeneration. They state that none of the lesions of the digestive tract, such as those associated with chronic coccidiosis, seemed related to the disease under consideration.

However, we question the carefulness of their autopsies, as they admit they were unable to find lymph-glands in any of their birds, and also may have missed lesions of the digestive tract, especially desquamation of the epithelium of the intestinal villi. In the examination of non-paralyzed birds these authors report lymphocytic infiltration of the nervous system in one-third of them. Injections of nerve tissue from paralysis cases produced paralysis in 25 per cent of their cases, in contrast to 7 per cent in the controls. They conclude that there is no correlation between coccidiosis and paralysis, and report 16 per cent of 60 cases of paralysis showed coccidia in sections, against 12 per cent in non-paralyzed birds. They state that there is little discrepancy between scrapings and stained sections, and that the absence of coccidia in stained sections is strong evidence against existence of coccidial infection. Because they found leucocytes of the lymphocytic type infiltrating nerve tissues, they suggest a new disease and a new name, *neurolymphomatosis gallinarum*.

Pugh[14] and, later, Perdrau and Pugh,[15] in examining the cerebro-spinal systems of dogs with the nervous form of distemper, found mononuclear infiltration, mostly lymphocytes, with varying proportion of plasma cells and a few polymorphonuclear cells.

Source of Material

The material used in this study has been collected from specimens received by the Department of Veterinary Pathology, College of Veterinary Medicine, Ohio State University, and by the Department of Veterinary Science and Bacteriology at the University of Wyoming. Total or partial paralysis of the legs existed in all the birds and animals used in this study except in the case of the controls. Blindness, total or partial, also existed in some of the birds. Many had gross changes in the nerve tissue and many did not. In most cases one or two birds were used from a flock with the occasional use of more, so as to obtain a fairly reliable representation of paralysis in poultry in general rather than in a few flocks. In order to study the lesions as they are found in the field, the birds were destroyed at the time received and were not held in order to attempt to produce or increase gross lesions in the nerve tissues. The tissues were collected immediately after death so as to guard against postmortem changes.

Experimental Work

As a routine procedure in every individual bird used in the study, the intestines, cerebrum, cerebellum, pons, medulla, optic lobe, and sciatic nerves were embedded, sectioned, stained and examined. Also these same tissues in every individual were run by Marchi's method for fatty degeneration. Various modifications of Marchi's method also were used. On most of the fowls a thorough autopsy was performed, scrapings were made from the intestines and, when negative for coccidia, the feces were centrifuged with sugar solution as originated by Sheather. Except in a few cases the intestines were washed, immersed in warm water and examined with low-power magnification for microscopic tapeworms. In individual cases sections were made from the spinal cord, dorsal root of spinal nerves, posterior or dorsal spinal ganglia, brachial plexus, median nerve, vagus nerve, liver, kidneys, lymph-glands, sclerocorneal junction of the eyeball, and the eyeball at the entrance of the optic nerve to the retina. Also in some cases serial sections were made from the brain in an attempt

to find infiltrations which single sections did not show. Enlarged nerve trunks, nerve plexuses, and dorsal nerve roots of spinal nerves with posterior ganglia were frozen and stained with Sudan III. Since sections of some enlarged nerves showed a clear, structureless substance in varying amounts between the infiltrating cells which resembled edema, the water content of the enlarged nerve tissue was determined and compared with normal.

In the case of the dogs and sheep, the microscopical examinations were made of the intestines, cerebrum, cerebellum, pons, medulla, optic lobe, gasserian ganglia, spinal cord, hippocampus, sciatic nerves, liver and kidneys.

In all, forty-seven cases of paralysis were studied; forty being chickens from twenty-eight flocks, five were dogs and two were sheep.

Discussion

Mallory,[16] in his text, "Principles of Pathologic Histology," says that toxic substances cause considerable infiltration with lymphocytes around the blood-vessels of nerve tissue and cause accumulations of some size in various tissues, and are most abundant after inflammation has lasted for weeks. He states also that the chief function of the lymphocytes in such infiltrations seems to be to neutralize or to dispose of substances which are being absorbed through the lymphatics.

The epithelial cells of the villi have certain selective action and govern to some extent what shall pass into the villi. When there is such extensive desquamation of this epithelium as was found in these cases with symptoms of paralysis, there is no such selective action, and by the ordinary processes of osmosis and diffusion, toxic products, most of which result from bacterial action, as well as bacteria themselves, pass from the intestinal contents into the villi. The resulting symptoms and lesions will depend upon the quantity, special affinity, and virulence of the toxic products that are absorbed in the villi divided by the resistance of the animal. When these toxic products are carried to the tissues they may cause leucocytic infiltration.

Catarrhal enteritis is very common in fowls and animals not showing paralysis and also is very common when they are showing no symptoms of disease. This does not mean that it is not the cause of paralysis, however, for we must apply the age-old maxim of pathology that disease always equals the virulence divided by the resistance.

From studies on the pathology of fowl paralysis we can see nothing that would warrant the recognition of it as a separate or distinct disease. A number of workers have failed to reproduce it by contact, injections and feeding; and also have failed culturally. The lymphocytic infiltration indicates the presence of toxic substances in the tissues and does not indicate a hereditary disease. Dogs with posterior paralysis show the same intestinal lesions and also the same cellular infiltrations of the brain. Perhaps paralysis cases in dogs and sheep do not show infiltrations of the peripheral nerves because they have not been of long enough duration. It is believed that extensive accumulation of lymphocytes into peripheral nerves is not a very acute process. The questions of species susceptibility and special affinity may enter into the explanation of this also.

In the studies on fowl paralysis in all stages of the disease, catarrhal enteritis, with very extensive desquamation of the epithelium lining the villi of the intestines, was the only consistent pathological lesion in each and every case.

The question of most practical importance is the cause of the catarrhal enteritis with such extensive desquamation. It is well known that catarrhal enteritis is caused by many things, and that one of the most common causes of desquamation of epithelium of the intestinal tract in chickens is coccidiosis. We admit there are cases of paralysis of fowls in which it is impossible to find coccidia and so probably they are not present. The dogs studied had paralysis but did not have coccidia. Some of them had tapeworms. This would strongly indicate that coccidia are not the only cause of paralysis. Rietz[17] found tapeworms in 26 birds out of a total of 36 that showed leg weakness, lameness, or paralysis. The sheep and some of the fowls and dogs examined did not have tapeworms or coccidia. This would suggest catarrhal enteritis of dietary origin as sometimes being the cause of paralysis.

Tyzzer's work shows that the development forms in chronic coccidiosis, which he has been able to produce experimentally with *Eimeria acervulina* and *E. maxima*, are for the most part limited to the epithelium of the villi, and that if reinfection does not take place the coccidia largely disappear from the digestive tract in ten days. This may account for absence of coccidia in some cases.

In our experience in fowl paralysis, it is frequently impossible to find coccidia by two or three scrapings from the digestive tract,

but by centrifuging the intestinal contents coccidia are found. Some claim that if coccidia are present to such an extent that they are causing serious trouble, several scrapings will show them. In acute cases this is no doubt true but in cases that have survived acute infection or harbor the species that cause chronic coccidiosis, the chances of missing the localized limited areas of accumulations of coccidia are very great. We have found them so localized that one villus of one section from a number of pieces of intestines embedded from the same fowl showed such localized accumulations in and around the epithelium. We feel this accounts for so many of our sections and scrapings being negative for coccidia, when the centrifuge showed their presence.

Tyzzer also found a species of coccidium for which he suggested the name *Eimeria mitis*, which was, experimentally, not virulent. However with lesions of severely desquamated epithelium, symptoms of paralysis, and the presence of coccidia, we feel the non-virulent species is not the only one that is, or has been, present.

Summary

The lesions were studied in forty-seven cases of paralysis, forty fowls from twenty-eight flocks, five dogs and two sheep.

The outstanding lesion, and the only consistent one in all the cases, was catarrhal enteritis with extensive desquamation of the epithelium of the villi of the intestines.

A leucocytic infiltration, principally with lymphocytes and endothelial cells, was found in thirty-two birds; twenty-five showing it in the sciatic nerves, fifteen in the cerebrum, fourteen in the medulla, eleven in the pons, nine in the optic lobe, six in the cerebellum, and six in the iris and the ciliary body. There were numerous other miscellaneous tissues showing infiltrations.

Of five dogs studied, four showed lymphocytic and endothelial infiltrations in the cerebro-spinal system, and one in the liver. Sciatic nerves of dogs and sheep contained no cellular infiltrations. One sheep had leucocytic infiltration of the cerebrum.

Thirteen fowls had distinct enlargements of the sciatic nerves. Four had a whitish opaque appearance to the eye. There were no enlargements of the nerves, or changes in the eyes in the dogs and sheep, which might be explained by acuteness of cases, species susceptibility, or specific affinity of toxic products.

Marchi's method and frozen sections stained with Sudan III showed slight fatty degeneration of some of the enlarged nerves.

The enlarged nerves were collected in a number of birds with paralysis from which microscopical studies were not made. These birds were not included in the total number. These enlarged nerves varied in their water content from 73 to 81 per cent, in comparison with 60 to 67 per cent in non-paralyzed birds which would indicate edema.

Twenty-four of the forty fowls had coccidia. Of the remaining birds six were not examined for coccidia other than by stained sections; in five the feces were not centrifuged; and in four others scrapings from the intestines were not examined.

Six fowls had tapeworms and seven were not examined for microscopic tapeworms.

Conclusions

It appears that fowl paralysis is not a specific disease but is the result of chronic catarrhal enteritis, with very extensive desquamation of the epithelium of the villi of the intestines and subsequent absorption of toxic products from the digestive tract.

There are a number of things that cause catarrhal enteritis. In fowls one common cause is coccidiosis. Other causes are intestinal parasites, especially tapeworms, and dietary disorders.

Acknowledgment

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Cinchona bark has been used in the treatment of malaria for three hundred years.

THE INCIDENCE AND TYPES OF DISEASE OF THE THYROID GLAND OF ADULT HORSES*

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A previous study[6] disclosed a high incidence of lesions of the thyroid gland in dogs. This suggested the possibility that other domesticated animals might likewise be affected, particularly animals that live for a relatively long period, such as dairy cattle and horses. The seriousness of certain types of diseases of the thyroid gland in man has long been recognized, and it may be that the usefulness of certain animals, horses especially, is terminated prematurely by this disease.

In order to investigate this subject further, the thyroid glands and other organs of from 100 consecutive horses on which necropsy was performed were studied. The animals were aged from two to twenty-eight years, and were destroyed because of various conditions which made them unfit for work.

In reviewing the literature, I failed to find reference to any extensive study of diseases of the thyroid gland in the adult horse; a few cases are cited in textbooks. Law[4] stated that goiter in horses may be evidenced by the enlargement of one or both lobes of the gland. He mentioned that Cadeac cited cases in which the goiter weighed as much as four pounds. Honert recorded a case of asphyxia in an adult horse due to pressure from a goiter. Massot, Neyrand and Truelsen observed difficult deglutition and dyspnea in a horse because of an enlarged thyroid gland. Cadiot and Jewsejenka observed what apparently was exophthalmic goiter in horses. Roder observed similar symptoms in a cow. These animals manifested weakness, palpitation, rapid pulse, enlargement of the thyroid gland, and exophthalmia. Johne described carcinoma of the thyroid gland of a horse accompanied by metastasis to the lungs. Dollar[2] reported a case of sarcoma of the thyroid gland of a horse. The neoplasm spread over the entire larynx during the period of a year and a half, finally causing death by suffocation. Lanzilatti removed from a horse an adenomatous left lobe of the thyroid gland about 10 cm. in

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diameter. The right lobe was small and was preserved. The horse was able to work a month after the operation.

Crocker[1] reported necropsy data concerning 768 horses. He mentioned lesions of the thyroid gland in two cases only; both were adenocarcinoma. The Sanitary Veterinary Service of Paris examined 39,800 horses and reported that 184 of these were affected with malignant tumor. It was not stated whether any of these tumors were of the thyroid gland.

The thyroid gland of the horse consists of two flattened, oval, lateral lobes and a narrow connecting isthmus. They are situated on and loosely attached to the trachea, immediately adjacent to the larynx.

The general condition, sex, age and weight of each of the 100 horses were noted carefully. All of the horses were shot and therefore all thyroid glands were obtained under the same conditions. Immediately after death, the glands were carefully dissected, weighed and examined grossly for superficial lesions. They were then placed in a 10 per cent solution of formaldehyde and allowed to become fixed. Later they were sectioned at 0.5-cm. intervals, so that the deep tissues of the gland could be observed. Both the normal tissue and the lesions were retained from each lobe for histologic study. Gross and microscopic examination of the glands permitted their being placed in four groups, namely, normal, hyperplastic, colloid and adenomatous.

Group 1: Thirty-four (34 per cent) of all the thyroid glands were classified in this group. This classification, however, is merely relative as some of the glands might have been placed in group 2. All glands showing marked hyperplasia, colloid degeneration and adenoma were excluded. Most of the acini of these glands contained colloid. However, there were some small cellular areas which were devoid of this substance (fig. 1). The acini were of varying sizes but without infoldings. The cells lining the acini were mostly low cuboidal and low columnar in type. The capsules of the glands and vessels were essentially normal. The average weight of the gland in this group was 23 gm. and represented 0.04 gm. of gland for each kilogram of body weight. This ratio ranged from 0.02 to 0.06 gm. of thyroid gland for each kilogram of body weight. It was observed that the latter figure was the highest obtained in a normal gland. All glands which showed a ratio of 0.07 gm. or more for each kilogram of body weight were abnormal on histologic examination.

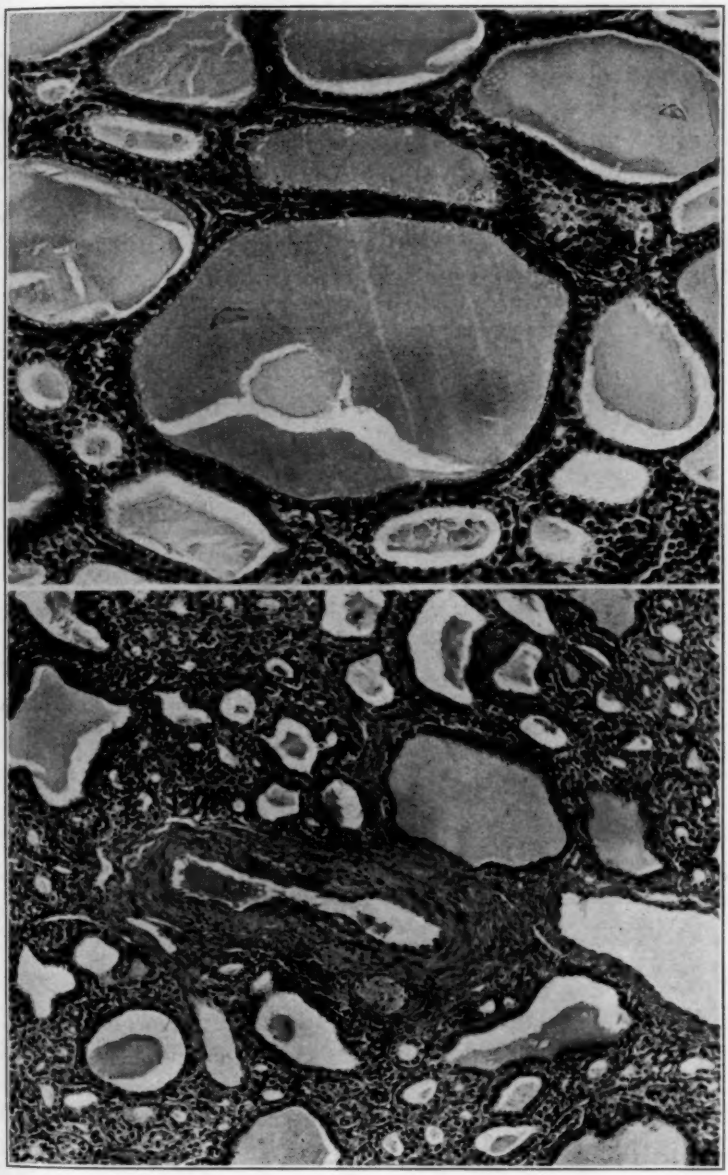

FIG. 1 (Above). Normal thyroid gland
FIG. 2 (Below). Hyperplastic thyroid gland. The tendency to infolding and hyperplasia of the acinar cells may be noted.

In this group, the average age of the horses was eighteen years. There were eleven males and twenty-three females. The average weight of the thyroid gland was 23.0 gm.

Group 2: In this group were twenty (20 per cent) of the glands studied. They ranged in size from 0.02 to 0.08 gm. of thyroid gland for each kilogram of body weight, the average ratio being 0.04 gm., the same as in group 1. Four glands weighed more than 0.06 gm. for each kilogram of body weight and might be classified as being both hyperplastic and hypertrophic.

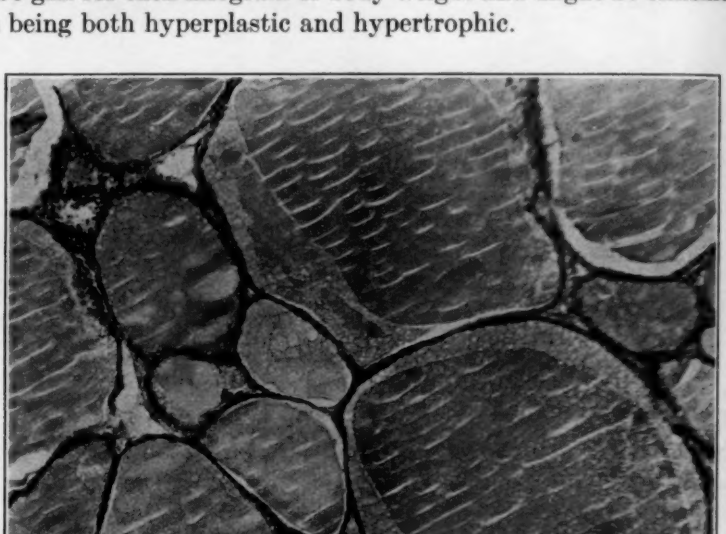

FIG. 3. Colloid thyroid gland. Absence of active cellular areas, acini filled with colloid, and flat to low cuboidal cells lining the acini are shown.

Histologically, all of the glands in this group were more than moderately hyperplastic. Eight contained large acini, with many infoldings or sprigs (fig. 2). These appeared to be very active. Most of the acinar cells were high columnar, containing large pale nuclei. These acini were devoid of colloid or contained poorly stained colloid. Four other glands, from 0.5 to 3 cm. in diameter, revealed large cysts filled with colloid material. The surrounding tissue in each of these glands was practically devoid of colloid and was very hyperplastic. The remaining eight glands contained little if any colloid, and were fetal in type. The cells were hyperplastic and practically filled the acini; the nuclei were

large, pale and granular staining. Most of the acini containing colloid were lined with high columnar epithelium. Thirteen of the thyroid glands had thickened capsules and fifteen had thickened or enlarged vessels. In five glands these features were not appreciably changed.

In this group, the average weight of the glands was 26 gm. The average weight of the horses was 555 kg., and the average age was eighteen years. There were nine females and eleven males.

Group 3: There were nine thyroid glands in this group. They ranged in size from 0.05 to 0.14 gm. for each kilogram of body weight, the average weight being 0.08 gm., which is 0.02 gm. above the maximal normal. Five glands were grossly enlarged,

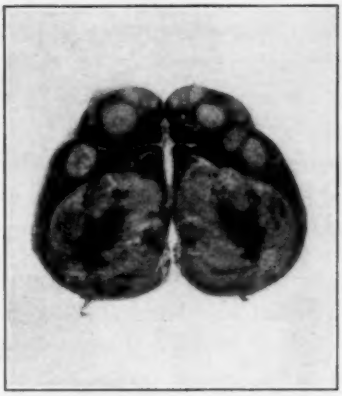

FIG. 4. Gross appearance of adenoma in the thyroid gland.

their size being 0.08 to 0.14 gm. for each kilogram of body weight. These could be classified as colloid goiters.

Histologically, the thyroid glands showed abnormal amounts of colloid. There were no cellular areas present, all acini being filled with colloid (fig. 3). The walls of the acini were thin and lined with flattened or low cuboidal cells having small dark-staining nuclei. The vessels of two of the glands were slightly enlarged; in none was the capsule appreciably thickened.

In this group, the average weight of the thyroid gland was 48 gm. The average weight of horses was 562 kg. and the average age was eighteen years. There were six females and three males.

Group 4: Thirty-seven (37 per cent) of the thyroid glands studied revealed adenomas. In thirty the adenomas were visible on gross examination (fig. 4). Eight glands were enlarged. Their

weight was 0.07 to 0.19 gm. for each kilogram of body weight. The average ratio for the entire group was 0.06 gm. for each kilogram of body weight.

Microscopically the adenomas differed slightly in structure. In three glands there was a papillary arrangement of cells (fig. 5). In one gland the adenoma was sharply circumscribed. The other two were more diffuse, they seemed to merge into the surrounding thyroid tissue, which was very hyperplastic, and numerous infoldings or sprigs were present. All three showed intra-adenomatous and extra-adenomatous hyperplasia. The glands were mottled on cross section.

The remaining thirty-four glands contained fetal and colloid types of adenoma (fig. 6), all of which were definitely circumscribed. Only thirty appeared to be partly or wholly encapsulated. Seventeen showed intra-adenomatous and extra-adenomatous hyperplasia, two showed intra-adenomatous and extra-adenomatous colloid degeneration and fifteen intra-adenomatous hyperplasia only.

In this group, the average weight of glands was 31.6 gm. The average weight of horses was 525 kg., and the average age eighteen years. There were twenty-one females and sixteen males.

Comment

This study reveals that lesions of the thyroid gland commonly exist in the horse. Sex is of no apparent significance as regards the incidence; age, however, may be a factor. The youngest animal in which adenoma was found was aged ten years. The oldest animal in the series was aged twenty-eight years and had a perfectly normal thyroid gland. The general condition of the animals in the various groups presents some interesting data. In the normal group, six animals (17 per cent), in the hyperplastic group, four (20 per cent), in the colloid group, one (11 per cent), and in the adenoma group, fourteen (approximately 40 per cent) were classified as being in poor flesh. The high incidence of loss of weight of the horses having adenomatous thyroid glands does not appear accidental, as these animals were all maintained under much the same conditions.

Just what the effect the adenomas may have had on impairment of health and function is merely speculation. It does not seem probable, however, that there was none. The vague ailments of horses so frequently attributed to a form of heart disease causing respiratory embarrassment and loss of flesh may be the direct

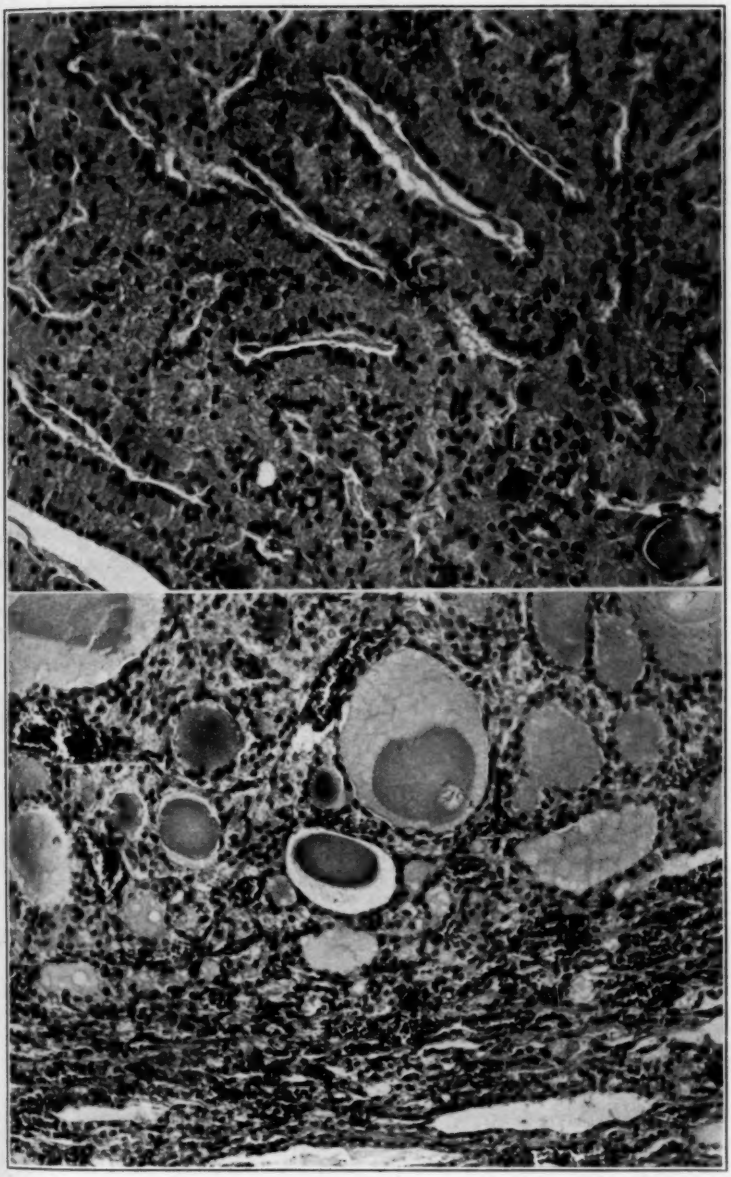

FIG. 5 (Above). Adenoma showing a papillary arrangement of cells.
FIG. 6 (Below). Fetal and colloid adenoma. The normal thyroid tissue is compressed at the margin causing a definite line of demarcation.

result of a hyperfunctioning thyroid gland. Some horses give shorter service than others, especially the race horse and hunter, some of which are unfit for active service while yet quite young. It may be that some of these horses are suffering from disease of the thyroid gland and could be placed back into service if the condition was recognized and proper treatment instituted. In this day, when many horses are valued $100,000 each, a knowledge of lesions of the thyroid gland of the horse may be of great economic importance.

Summary

The thyroid glands of 100 horses were studied grossly and microscopically. The data obtained indicate that any gland having a weight greater than 0.66 gm. for each kilogram of body weight is abnormal. However, smaller glands may also show pathologic changes.

Of 100 thyroid glands studied, thirty-four were classified as normal, twenty as hyperplastic, nine as colloid and thirty-seven as adenomatous. The adenomas consisted of benign papillary, fetal and colloid; none was malignant.

Sex apparently is of no significance. Age, however, should be considered, as the youngest animal having an ademomatous gland was aged ten years.

Approximately 40 per cent of horses having adenomatous thyroid glands were in poor general condition, whereas only 17 per cent in the three other groups were thus classified. This may indicate that some adenomas impair the health of the horse.

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Course on Exotic Pathology and Parasitology

A course on Exotic Pathology and Parasitology will be held in Berlin, Germany, at the Pathological Museum of the Charity Hospital, beginning March 5, 1931. The lecturers giving this course are Professors Bruhns, Nöller, Claus Schilling and Ziemann, according to information kindly sent us by Dr. Karl F. Meyer, of San Francisco.

A COMPARISON OF THE INTRADERMAL AND AGGLUTINATION TESTS FOR PULLORUM DISEASE, BASED ON DEMONSTRATING THE INFECTION IN THE HATCH FROM REACTORS*

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The occurrence of pullorum disease in chicks can usually be traced directly to the presence of infected hens in breeding flocks. It is generally accepted that the infection is transmitted from the ovary of the hen, through the egg, to the chick, and that widespread dissemination occurs in incubators by means of the down of chicks from infected eggs. The elimination of infected hens from the breeding stock is at this time the most effective and practical method of preventing the disease.

The value of the agglutination test for detecting carriers of *Salmonella pullorum* infection in fowls was first reported by Jones,[1] in 1913, and is now used extensively in some sections of the United States. An intradermal or intracutaneous test was first applied to pullorum disease by Ward and Gallagher,[2] in 1917. The procedure that is required to perform the agglutination test makes it rather expensive and probably has been an important factor in retarding its more general use for the eradication of pullorum disease. The increasing interest in the intradermal or pullorin test has been stimulated by the demand for a test that is simpler in application and therefore less expensive than the agglutination test.

Many investigations have been made for the purpose of determining the accuracy of the agglutination and intradermal tests as a means of detecting *S. pullorum* carriers in breeding fowls.

Ward and Gallagher[2] reported that there was complete agreement between the intradermal and agglutination tests and autopsy, in 70 per cent of the cases. The absolute disagreements were very small, which indicated that the two tests were about equally reliable.

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Scherago and Benson[3] compared the intradermal with the agglutination test and concluded that the intradermal test was of little value as a diagnostic agent.

Fuller[4] stated that the intradermal test detected a large percentage of infected fowls, but did not detect all in a heavily infected flock, and that only a very small perentage of non-infected fowls reacted to the test.

Graham and Tunnicliff[5] suggested the possible value of pullorin in diagnosing pullorum disease in mature fowls and based their conclusions on the results obtained by the intradermal and agglutination tests, and by autopsy.

Graham, Tunnicliff and McCulloch[6] reported that the intradermic test was almost as consistent in demonstrating the presence or absence of infection as the agglutination test and suggested that the discrepancies between the two tests, as judged by diseased ovaries and bacteriological findings, may be due to the personal factor involved. Neither the intradermal test nor the agglutination test was considered a perfected procedure, but the best means available to eliminate carriers from breeding stock.

Edwards and Hull[7] concluded from their data that the agglutination test is much more reliable than the intradermal test in detecting fowls infected with pullorum disease.

Gwatkin[8,9] checked the two tests by autopsies and found the agglutination test more satisfactory than the intradermal test for detecting carriers of *S. pullorum*.

Stafseth and Thorp[10] reported that the intradermal test was decidedly inferior to the agglutination test in regard to its ability to detect carriers of pullorum disease.

Bushnell[11,12,13] concluded that the intradermal test cannot be recommended as a substitute for the agglutination test in eliminating fowls infected with *S. pullorum*, but that it appears to have enough merit to justify further investigation.

B. A. Beach[14] investigated the comparative value of the intradermal and agglutination tests, and seemed to conclude that the tests were too inaccurate to be recommended for control measures.

J. R. Beach[15] reported that in some flocks there is a rather close correlation between the intradermal and agglutination tests, and the results of bacteriological examinations of the fowls. The intradermal test did not appear to be so satisfactory in detecting carriers of *S. pullorum* as the agglutination test.

Michael and Beach[16] suggested the possibility of developing a type of pullorin that might be of value for the diagnosis of pullorum disease.

Rettger, McAlpine and Warner[17] obtained a high degree of correlation between the agglutination test and postmortem observations. The intradermal test gave results which frequently failed to agree with the agglutination test. Such discrepancies occurred most often in the so-called negative groups, and were so marked as to render the method as applied useless.

From the literature cited it is evident that the results of the agglutination test have been used as the principal criterion of the value of the intradermal test. Many investigators have attempted to determine the merits of the two tests by checking the reactions with postmortem examinations and bacteriological findings. Owing to the variable results reported by the different investigators as to the accuracy of intradermal and agglutination tests in detecting carriers of *S. pullorum*, the writers undertook a study of the comparative efficacy of the two tests, basing the comparison on demonstrating infection in the hatch.

Intradermal and Agglutination Methods

The intradermal fluid, now commonly called pullorin, used in these tests, was prepared by growing five strains of *S. pullorum* on nutrient agar for three days. The organisms had been isolated from chicks and mature fowls. The growth was harvested with phenol-salt solution, filtered through paper and washed twice. The heavy-cell sediment was suspended in N/10 NaOH for a half-hour, and then diluted to ten times its volume with phenol-salt solution. The pullorin had a H-ion concentration of pH 8 and was tested for sterility before being used. In making the tests about 0.05 cc of this material was introduced into the skin of the wattle. Observations were made at the 18th and 24th hours following the injection. Edematous swellings of the wattle which persisted for 24 hours were considered positive reactions.

Three dilutions (1:25, 1:50 and 1:100) of the serum were employed in the agglutination test. The antigen was prepared from the five strains of *S. pullorum* used in making the pullorin previously mentioned. The reactions were considered positive when agglutination was complete in the 1:25 dilution.

Experiment

A flock consisting of 201 Rhode Island Red fowls was selected for this investigation. Pullorum disease was known to exist in

the group, as the infection had been demonstrated in chicks hatched from eggs produced by hens in this flock. All the fowls on the farm were subjected to the intradermal and agglutination tests. Twenty-four birds reacted to both tests. Twenty reacted to the agglutination test only, and 27 to the intradermal test only. All reacting birds were removed from the flock and grouped according to their reactions into:

Group I: Reactors to both the agglutination and intradermal tests.

Group II: Reactors to the agglutination test only.

Group III: Reactors to the intradermal test only.

The three groups were placed in separate houses, provided with wire "sun-parlors." All the eggs produced by these hens during the period of the experiment, regardless of size or shape, were used for hatching. The eggs from each group were hatched in separate incubators and the chicks brooded for a period of one month in separate brooders. Ten different hatches were obtained from each group during the period of the experiment. The number of eggs incubated, the number of chicks hatched and the mortality of each group are given in table I.

A study of table I shows that from eggs produced by the 24 hens reacting to both the intradermal and agglutination tests,

TABLE I—*Eggs incubated, chicks hatched and mortality in three groups*

	GROUP I. EGGS FROM HENS REACTING TO BOTH TESTS			GROUP II. EGGS FROM HENS REACTING TO AGGLUTINATION TEST ONLY			GROUP III. EGGS FROM HENS REACTING TO PULLORIN TEST ONLY		
HATCH	EGGS	CHICKS	DEATHS	EGGS	CHICKS	DEATHS	EGGS	CHICKS	DEATHS
A	63	28	25	57	18	18	75	39	1
B	102	29	26	98	32	25	115	43	0
C	109	49	15	108	36	31	125	52	1
D	75	31	15	60	30	25	91	34	2
E	73	31	10	55	36	15	111	67	0
G	65	25	3	52	33	9	92	53	1
H	71	20	0	58	29	15	94	31	1
I	51	27	19	47	29	11	95	51	4
J	57	23	4	34	13	12	79	31	1
K	61	26	9	35	18	5	65	30	0
Totals	727	289	126	604	274	166	942	431	11
Per cent		39.6	43.5		45.3	60.5		45.7	2.5

39.6 per cent hatched, and a mortality of 43.5 per cent occurred in the chicks. The eggs of the 20 hens reacting to the agglutination test and not to the intradermal test had a hatchability of 45.3 per cent and a chick mortality of 60.5 per cent. Eggs from the 27 hens that reacted to the intradermal and not to the agglutination test showed a hatchability of 45.7 per cent and a chick mortality of 2.5 per cent.

No deaths occurred in the seventh hatch of group I nor in the second, fifth and last hatches of group III.

Most of the chicks that died during the brooding period were examined to determine whether or not they were infected with *S. pullorum*. Blood from the heart of these chicks was placed on a chicken-agar medium and cultures thus secured were used in agglutination tests with known positive and negative pullorum serums. The cultures were further identified by their reactions in media containing dextrose, maltose, lactose and sucrose with brom-thymol-blue as the indicator. The results of the bacteriological examinations of cultures isolated from the chicks that died are given in table II.

The data presented in table II show that *S. pullorum* was isolated from 95.6 per cent of the chicks examined in group I, and from 88.1 per cent of the chicks in group II. Group III had a mortality of 2.5 per cent, but no culture of *S. pullorum* was obtained from these chicks.

TABLE II—*Bacteriological examinations of chicks that died*

	GROUP I			GROUP II			GROUP III		
HATCH	CHICKS	POS.	NEG.	CHICKS	POS.	NEG.	CHICKS	POS.	NEG.
A	12	10	2	12	12	0	1	0	1
B	19	19	0	19	15	4	0	0	0
C	10	10	0	22	22	0	1	0	1
D	16	16	0	16	15	1	2*	0	0
E	8	7	1	14	12	2	0	0	0
G	4	4	0	8	4	4	1	0	1
H	0	0	0	17	13	4	0	0	0
I	8	8	0	6	6	0	1	0	1
J	3	2	1	4	4	0	1*	0	0
K	12	12	0	9	9	0	0	0	0
Totals	92	88	4	127	112	15	7	0	4
Per cent		95.6			88.1		2.5		

*No culture.

The Negative Flock

The 130 fowls that did not react to the intradermal and agglutination tests were isolated from all other poultry and used for breeders. During the experiment, 1730 chicks were hatched from 2400 eggs produced by the negative flock. The incubator in which these chicks were hatched was used only for eggs from this flock. A few chicks died in this group, but *S. pullorum* was not isolated from these birds.

Discussion and Conclusions

In this experiment it was determined that hens in the group reacting to the agglutination test but *not* to the intradermal test, transmitted *S. pullorum* infection in each of ten hatches. If the flock had been divided on the basis of the intradermal test only, these hens would have remained in the negative group and would have been a source of disseminating pullorum disease.

The hens reacting to the intradermal test but not to the agglutination test, failed to transmit the infection to chicks during the hatching period.

These observations indicate that the intradermal test, under the conditions of this experiment, was not so satisfactory as the agglutination test in detecting carriers of pullorum disease in mature fowls.

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STUDIES UPON ASTRAGALUS CAMPESTRIS, A POISON PLANT*

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After stockmen of southwestern Wyoming and northern Colorado had suffered considerable loss of cattle and sheep through a disease locally known as "cracker heel," it was learned that animals which had grazed upon a milk vetch in the forests were the most commonly affected. Sheep frequently succumbed to pneumonia but cattle showed an incoördination of the hind legs. The disease has been attributed to *Astragalus campestris*, a close relative of the loco plants.

In British Columbia, Canada, Dr. E. A. Bruce[1] has conducted feeding experiments in order to secure all data possible upon the cause and symptoms of a somewhat similar disease. After a few experiments Bruce became convinced that a plant, also identified as *Astragalus campestris*, was responsible for the trouble.

Bruce found that lactating ewes and cows were more often affected by the ingestion of this plant than were males and non-lactating females. Suckling calves and lambs were not affected. Bruce classified the resulting illnesses into two types: the incoördination type, which caused the animal to be unsteady upon its feet or unable to stand at all, and the laryngeal type, which accompanied a greater or less paralysis of the respiratory organs. Horses affected with the laryngeal type roar during expiration. The periods of the incoördination type were intermittent and might be accompanied by loss of fetlock control, so that the animal "knuckled over" or dragged its feet.

Sheep exhibited dyspnea after exertion, a protruding and cyanotic tongue, and a wheezing or roaring sound. Loss of voice, a cough, grinding of the teeth, and slight bloating also were noted. Pain or fever was infrequent except during dyspnea, but there was considerable difficulty in swallowing because of paralysis. Thus, small particles of food were frequently allowed to enter the lungs and caused foreign-body pneumonia. The animals lost weight despite their good appetites.

Cattle showed symptoms similar to those of sheep. Insalivation was observed as was also bladder irritation. When arising,

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The Negative Flock

The 130 fowls that did not react to the intradermal and agglutination tests were isolated from all other poultry and used for breeders. During the experiment, 1730 chicks were hatched from 2400 eggs produced by the negative flock. The incubator in which these chicks were hatched was used only for eggs from this flock. A few chicks died in this group, but *S. pullorum* was not isolated from these birds.

Discussion and Conclusions

In this experiment it was determined that hens in the group reacting to the agglutination test but *not* to the intradermal test, transmitted *S. pullorum* infection in each of ten hatches. If the flock had been divided on the basis of the intradermal test only, these hens would have remained in the negative group and would have been a source of disseminating pullorum disease.

The hens reacting to the intradermal test but not to the agglutination test, failed to transmit the infection to chicks during the hatching period.

These observations indicate that the intradermal test, under the conditions of this experiment, was not so satisfactory as the agglutination test in detecting carriers of pullorum disease in mature fowls.

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STUDIES UPON ASTRAGALUS CAMPESTRIS, A POISON PLANT*

By ALVAH R. MCLAUGHLIN, *Laramie, Wyoming*

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After stockmen of southwestern Wyoming and northern Colorado had suffered considerable loss of cattle and sheep through a disease locally known as "cracker heel," it was learned that animals which had grazed upon a milk vetch in the forests were the most commonly affected. Sheep frequently succumbed to pneumonia but cattle showed an incoördination of the hind legs. The disease has been attributed to *Astragalus campestris*, a close relative of the loco plants.

In British Columbia, Canada, Dr. E. A. Bruce[1] has conducted feeding experiments in order to secure all data possible upon the cause and symptoms of a somewhat similar disease. After a few experiments Bruce became convinced that a plant, also identified as *Astragalus campestris*, was responsible for the trouble.

Bruce found that lactating ewes and cows were more often affected by the ingestion of this plant than were males and non-lactating females. Suckling calves and lambs were not affected. Bruce classified the resulting illnesses into two types: the incoördination type, which caused the animal to be unsteady upon its feet or unable to stand at all, and the laryngeal type, which accompanied a greater or less paralysis of the respiratory organs. Horses affected with the laryngeal type roar during expiration. The periods of the incoördination type were intermittent and might be accompanied by loss of fetlock control, so that the animal "knuckled over" or dragged its feet.

Sheep exhibited dyspnea after exertion, a protruding and cyanotic tongue, and a wheezing or roaring sound. Loss of voice, a cough, grinding of the teeth, and slight bloating also were noted. Pain or fever was infrequent except during dyspnea, but there was considerable difficulty in swallowing because of paralysis. Thus, small particles of food were frequently allowed to enter the lungs and caused foreign-body pneumonia. The animals lost weight despite their good appetites.

Cattle showed symptoms similar to those of sheep. Insalivation was observed as was also bladder irritation. When arising,

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cattle showed loss of control of the hind legs and often dragged them for several yards. One of Bruce's collaborators noted that three cows were affected with temporary amaurosis from which two recovered in a few hours, but one remained blind for two days.

Many of these animals, if removed to other feed, recovered. Others, after a lapse of several months, died of secondary pneumonia. Upon autopsy Bruce found lesions characteristic of broncho-pneumonia, cardiac failure, or anemia. The heart was flabby, there was increased pericardial fluid, and often pericarditis. There were some evidences of nephritis.

Experimental Work

In order to make a laboratory study of the conditions produced by this plant, quantities of it were collected in the pre-bloom, flowering, and seeding stages. This material was cleaned, dried, and ground for percolation. Preliminary studies showed that the active principles of the plant were capable of extraction by means of 90 per cent alcohol; but if the alcohol were evaporated by means of gentle heat, the active constituents could then be taken up in 20 to 50 per cent alcohol.

An extract of the flowering stage of the plant in 50 per cent alcohol was given to each of four rabbits in 30-cc amounts by means of the stomach-tube, every other day, over a period of time. The first rabbit died on the second day. The only postmortem lesion found was congestion of the kidneys. The second rabbit survived until the fifteenth day, when he died following a dose of 48 cc of the extract. The visible postmortem lesions noted were catarrhal gastritis and enteritis. The third rabbit showed the same history as the second and likewise died on the fifteenth day after an excessive dose of the extract. Small necrotic areas upon the kidneys were found in addition to the catarrhal gastro-enteritis seen in the second rabbit. The fourth rabbit became very ill following the second dose of the extract and thereafter received but half of the original amount of the extract. He showed no further ill effects. A notable effect of this extract upon rabbits was the excretion of urine which upon exposure to the air became dark brown or black in a short time.

The major portion of the material of the seeding stages of the plant was treated in the manner described above and then taken up in 20 per cent alcohol. During the course of 13 days, two rabbits, one weighing 1,600 grams, and another weighing 1,175

grams, were given each a quantity of this solution equivalent to one-fourth of the body weight, if expressed in terms of air-dried material. This plant, freshly gathered, contains 80 per cent moisture. When these animals were anesthetized and examined, both showed catarrhal gastritis and the 1,600-gram rabbit showed a few hemorrhagic areas on the lungs.

The remaining percolate from the seeding stage was freed of most of its resin and then treated with basic subacetate of lead. After filtering, the lead in solution was removed by means of hydrogen sulphid and filtered off. The filtrate was then evaporated to a semi-solid syrupy mass, each gram of which represented 123 grams of air-dried plant material.

Four rabbits were given each 150 mg. of this material daily. The first rabbit developed "sore eyes" on the sixth day and died during the night after the eleventh treatment with the extract. On the fourteenth day, the second rabbit showed coryza and died on the sixteenth day. The third rabbit exhibited slight evidences of coryza and sore eyes on the twentieth day and was etherized and destroyed. The fourth rabbit, although in apparent good health when treated on the morning of the twenty-third day, was found dead in the afternoon.

Autopsies revealed necrotic areas on the liver of the first rabbit, gastric ulcers and pneumonia in the second rabbit, punctate hemorrhagic areas, 1 to 2 mm. in diameter, upon all of the lobes of the lungs and small necrotic areas upon the kidneys of the third rabbit, and severe pleuro-pneumonia and pericarditis in the fourth rabbit. It is surprising that this last rabbit survived so long.

While undergoing the experiments, these animals were well housed and carefully fed. One of the controls likewise developed "sore eyes." All rabbits showing such symptoms were given instillations of a solution of boric acid. The suppuration of the eyes of the control rabbit yielded to the boric acid treatment.

In order to determine if any constituent of the above extract were capable of causing a loco effect, three-month-old puppies were daily given 0.5 gram of the syrupy material for three weeks, then one gram for a week, following which one of the puppies was etherized and a postmortem examination made. An intense congestion of the lungs was found, as were also evidences of a slight nephritis. The second puppy was fed 1.5 grams of the syrupy material for another week and was then destroyed. This dog likewise showed evidences of nephritis. Only a small portion

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of the lungs was found congested and that showed beginning resolution. In another week, it probably would have been completely cleared. These dogs, while living, showed no visible abnormal symptoms.

Animals were also anesthetized and connected with the instruments regularly used in the laboratory for making kymographic records of respiratory and blood-pressure changes. When small amounts of the extract of the flowering stage of *Astragalus campestris* were injected intravenously, there occurred intermittent or periodic alterations of respiration. In the case of a cat, these intermittent periods of respiration were quite similar to those generally described as the Cheyne-Stokes type. Very marked alterations in blood-pressure and heart-rate accompanied the periods of respiratory activity. Dogs and sheep exhibited periodic alterations of respiration accompanied by slight depression of blood-pressure, but not to such a marked degree as shown by the cat. Although several rabbits were employed, no noteworthy effects were produced on these animals.

Conclusion

Since the daily feeding of an especially prepared extract—filtrate from treatment with subacetate of lead—to rabbits and to dogs resulted in congestion of the lungs and pneumonia in the former, the writer believes that this plant, *Astragalus campestris* contains a substance capable of injuring the walls of the capillaries, thereby altering their permeability. This altered permeability is more readily apparent in the delicate tissues of the lungs than elsewhere in the animal body.

In the case of rabbits and probably also in sheep, ever-present bacterial organisms invaded the scene and produced the picture recorded. Dogs, because of a much higher resistance and the warmer weather, survived this congestion without revealing any external symptoms. It is probable also that this or another substance is responsible for the injury to the kidneys thereby causing the slight nephritis noted.

The fact that intravenous injection of extracts of this plant in cats, dogs and sheep prepared for making kymographic records of blood-pressure and respiration cause intermittent respiration and even death by respiratory inhibition, shows it to have a powerful depressant action upon the respiratory center.

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LUNG FLUKES OF THE GENUS PARAGONIMUS IN AMERICAN MINK

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In the course of studies on the animal parasites of the American mink, *Lutreola vison*, it was found that a lung fluke of the genus Paragonimus is not uncommon and is apparently widely distributed. Since it appears to be the most dangerous of the several flukes infecting this important fur-bearer, a preliminary report seems advisable.

It would appear from my observations that mink constitute the normal hosts of this parasite in this country and hence afford the most available source for life history studies that are fundamental to control measures. These studies are being undertaken cooperatively by the Division of Entomology and Economic Zoology and the Department of Zoology of the University of Minnesota.

It is a pleasure to express my appreciation to Dr. W. A. Riley for the suggestion of the problem and for his constructive criticisms and suggestions during the course of the work. I wish to thank him also for specimens of lung flukes from the cat from his collection and for specimens from the pig which he obtained, through the kindness of Dr. M. C. Hall, from the collection of the U. S. Bureau of Animal Industry.

The Asiatic lung fluke, *Paragonimus westermanni*, has long been recognized as a dangerous parasite of man in the Orient. The fact that this or a closely related species occurs in the United States has been known since 1894, when Ward[12] identified specimens from a cat at Ann Arbor, Michigan, as belonging to this species. The evidence that the parasite was endemic in this country was not conclusive, as the history of the cat was unknown.

Subsequently, however, flukes of this genus were found occasionally in the cat, dog, pig and goat in this country. Kellicott[7] found it in the dog at Columbus, Ohio, while Stiles and Hassall[11] reported that it was found in the course of slaughter-house inspection of hogs at Cincinnati. Null[10] mentioned its presence in cats

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and dogs in San Francisco. Nickerson[9] and Hanson[4] recorded its presence in the cat in Minneapolis and Wisconsin, respectively. Dr. W. A. Riley found it in the dog at Ithaca, New York, and again in the cat in Minneapolis (unpublished records). Hall[3] reported the only known case of Paragonimus from the goat. Feldman and Essex[1] found the lung fluke in a cat at Rochester, Minnesota, and made a careful pathological study of the lungs. In addition to these records, Paragonimus has been found repeatedly in pigs by government inspectors, principally at Cincinnati.

In 1908, Ward[13] announced that the American lung fluke was not *Paragonimus westermanni* but a new species which he named *Paragonimus kellicotti*. Detailed studies and comparisons of the morphology of this and the Asiatic species were presented by Ward and Hirsch.[14]

Strangely enough the initial discovery of lung flukes in American mink was made in France. In 1927, a large shipment of mink was sent from Minnesota to Europe to stock fur-farms there. Henry[6] made postmortem examinations of two animals. He found the lungs of both heavily infested with lung flukes which he identified as *Paragonimus kellicotti*.

Freund[2] reported a case of lung fluke from an American mink in Germany. Although he did not attempt to name the parasite it was undoubtedly Paragonimus.

My findings were made in mink which died on various fur-farms in Minnesota during the winter of 1929-30. Eighty-four carcasses were examined and 7 of these (8.09 per cent) were infected with Paragonimus. Examination of 150 random fecal samples by the direct smear method showed a 6.33 per cent incidence of this parasite.

With the exception of the remains of encysted individuals in the body cavity, and a few living ones free in the thorax, all the lung flukes were found in cyst-like cavities in the lungs (fig. 1.) These cysts were so large in proportion to the lungs that they appeared as bulbous projections on the surface, the larger ones being visible from both sides. The outside of the cysts is a slate-blue color which contrasts markedly with the pink of the lungs. While in other hosts the lung fluke cysts contain two parasites, in mink they often contain several; in one case as many as six. The photograph (fig. 1) is of a pair of heavily infested lungs.

Occasionally there were found slate-colored growths in various places in the body cavity. As some of these contained Paragonimus eggs they were considered flukes, which had died and become encased in connective tissue. Musgrave[8] found similar "mummified" flukes in human cases in the Philippines.

Henry, in his cases, found that the lungs of one animal contained a total of seven cysts and those of the other ten. Because of the number of parasites in one host and because some cysts contained several individuals, Henry suggested that the parasites

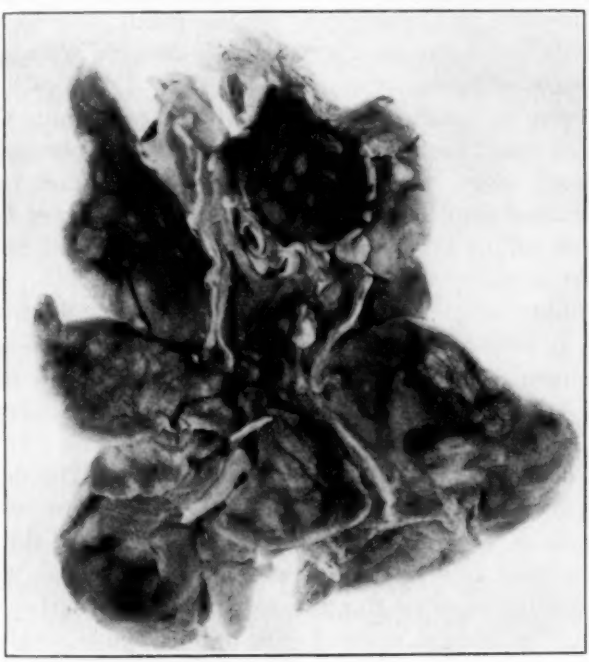

FIG. 1 Dorsal view of mink lungs infested with Paragonimus. One cyst is opened showing six parasites inside.

multiplied in the definitive host. This hypothesis is hardly tenable in view of our knowledge of the life histories of trematodes in general.

The cysts in the lungs contain, in addition to the parasites, a dark brown, slimy fluid whose color is derived principally from large numbers of fluke eggs. The cysts open into bronchioles which act as outlets for the eggs and excretory waste of the parasites. This brown exudate is found in the trachea in severe cases, sometimes in large quantities. It is swallowed and the

lung fluke eggs as well as the eggs of intestinal parasites are found in the feces.

Feldman and Essex, in a pathological study of a case of paragonimiasis in a cat, found that large parts of the lungs, not occupied by the parasites themselves, were filled with eggs which caused an inflammation and collection of white corpuscles. Many of the smaller bronchioles were so filled with débris and eggs that they were completely closed to the passage of air. Most of these eggs were in some stage of decomposition and some of the shells contained phagocytes. The injury to the lungs in heavy infections is tremendous, as the small amount of lung tissue left between the cavities is largely clogged with eggs and waste of various kinds.

Head,[5] who was obviously dealing with Paragonimus although he refers to it as "the" liver fluke, states that "a peculiar observation that I make, however, is that the flukes attack the lungs in mink rather than the livers and locate themselves first just at the base of the lung, spreading with growth until finally the major part of one or both lungs are destroyed."

Lung flukes are large, thick trematodes, varying from 6 to 15.5 mm. in length and from 3 to 7 mm. in width. When alive they assume a great variety of shapes so the form is by no means constant. A typical specimen, however, is widest about one-third of the length back from the anterior end. Often the ends are drawn out into knob-like projections. The dorsum is arched while the venter is nearly flat. There are two easily visible suckers, one at the anterior end and one on the ventral side. The color is a light gray with a reddish tinge, while the dark, branching vitelline glands show through the surface around the sides. The body is covered with minute spines which can be seen only with the microscope.

Detailed comparisons of the mink parasites with Ward and Hirsch's description of *Paragonimus kellicotti* and with specimens from the cat and pig left no doubt that they were of the same species. As the cuticular spines are the only morphological features used in determining species of this genus, they were subjected to careful study. A rather wide variation in the form of these was found, so further work is being carried on to determine their significance.

Diagnosis is most easily made by fecal examination. In most cases the ova are so numerous that they are readily detected by the use of the direct smear method. Technics involving the flota-

tion of ova in a solution of high specific gravity are generally not effective for trematode eggs, as the solution enters the operecula and the eggs sink. Sedimentation or centrifuging in water is satisfactory. The eggs (fig. 2) are distinctive in appearance and can be identified by the prominent operculum and the thickening of the shell at the opposite end. They vary from .091 mm. to .073 mm. in length and from .06 mm. to .048 mm. in width, averaging .078 mm. x .056 mm.

The life history of the Asiatic lung fluke, *Paragonimus westermanni*, has been demonstrated. The ova develop in water and in from three to six weeks motile miracidia hatch from them. These enter snails of the genus Melania. After a period of development in this host, the cercariae are freed and enter the fresh water crabs, Potamon and Eriocheir, or the fresh water crayfish,

FIG. 2 Egg of Paragonimus from mink. x 500.

Cambaroides. The definitive host becomes infected by eating these crustaceans or, more rarely, by drinking water which has harbored them.

Paragonimus kellicotti, like its Asiatic congener, uses the crayfish as second intermediate host. We have succeeded in heavily infecting cats with cysts from the pericardial region of *Cambarus immunis spinirostris*. This being the case, there is little chance for pen-raised animals to become infected, as they are seldom fed these crustaceans. None of the cases reported have been in animals definitely known to be pen-raised. Furthermore, subsequent examinations of 143 carcasses, nearly all young animals bred in domestication, revealed no cases of Paragonimus. For this reason it would be well for mink-buyers to insist on strictly pen-raised stock and to have fecal examinations made to determine possible infections.

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Bulletin Describes Poultry Diseases

Preventive measures properly applied constitute the best means of controlling poultry diseases and parasites, according to a bulletin, "Diseases and Parasites of Poultry," just issued by the U. S. Department of Agriculture.

Among the precautionary measures mentioned are the immediate separation of sick birds from healthy flocks, frequent removal of droppings, sanitation of feed and water utensils, and the use of clean soil in runways for both young and old birds. Good disinfectants destroy the germs of contagious diseases, parasites such as mites, and in some cases the eggs of parasitic worms. As the most severe damage from parasites occurs among young birds, special care should be taken to protect them from infection.

Among the most serious poultry diseases described in the bulletin are bacillary white diarrhea of young chicks, and avian tuberculosis which affects principally old birds. Coccidiosis among young chicks and blackhead of turkeys are mentioned as important parasitic diseases. Lice, mites and worms take a heavy toll in many flocks. The publication discusses ailments such as roup, colds, bronchitis, and others resulting from exposure.

The new bulletin is a revision of and supersedes a former bulletin entitled, "Diseases of Poultry." It may be obtained on application to the Office of Information, U. S. Department of Agriculture, Washington, D. C.

Scientific evidence appears to be accumulating to indicate that the common cold is caused by a filtrable virus.

RECENT ADVANCES IN THE PHYSIOLOGY OF DIGESTION

IV. The Intestine*

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Control of Pancreatic Secretion

That the initial flow of pancreatic juice is caused by the stimulus of eating and is under nervous control is generally recognized. That a chemical excitant (hormone) is also concerned has been widely believed since the classical work of Bayliss and Starling. However, in late years, opinion has been divided on the question whether the mechanism is a normal one or simply one that may be elicited under certain experimental conditions. Recent evidence fully confirms the earlier views to the extent of showing that a hormone mechanism may be concerned in pancreatic secretion, and the preponderance of evidence favors the view that the mechanism is normal. Ivy and co-workers[1,2] have shown that a piece of pancreas transplanted under the skin secretes after the animal is fed. This is proof of a humoral mechanism. That the humoral agent is a hormone is made practically certain by an experiment in which a piece of intestine was transplanted under the skin, and later a pancreatic transplant was made. When a weak hydrochloric acid solution was applied to the mucous membrane of the piece of intestine, the pancreatic transplant would secrete. Necheles and Lim[3] have made a valuable contribution to our knowledge of the pancreatic secretory excitant. They demonstrated that it could be recovered from the circulating blood of dogs by vividialysis, and that when it was injected into the blood-stream of other dogs, the pancreas of the latter would secrete. It was further found that the amount of the excitant in the blood could be increased by feeding or by injecting hydrochloric acid into the duodenum.

Bayliss and Starling held that the pancreatic secretory excitant, which they termed secretin, exists in the duodenum in the form of prosecretin, which is converted to secretin by hydrochloric acid. Recent work by Mellanby[4] seems to show that secretin

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exists in the intestine in the active state and, therefore, requires no activation. He further found that secretin possesses the properties of a polypeptide, and that the purified product has no depressor effect on blood-pressure. Mellanby studied the distribution of secretin and found that it varies considerably in different animals. In the cat it is found practically only in the duodenum. In the pig considerable amounts occur at least ten feet from the pylorus. In the goat large quantities are found not only in the duodenum but also in the ileum.

Duodenum

Duodenal glands: The distribution of the duodenal glands varies greatly in different species.[5] The function of these glands is not known, although the work of Bergman, Dukes and Yarborough,[6] in which extracts of the gland substance of domestic animals were studied for enzymic action, suggests that they produce an amylolytic enzyme. Definite conclusions could not be reached because it was impossible to be certain that the extracts were not contaminated by amylase from blood or lymph. No lipase or protease was found.

Effect of removal of the duodenum: It has been believed by some workers that the duodenum possesses special functions necessary for the animal's well-being, or even that it is necessary for life. The effect of duodenectomy was studied by Mann and Kawamura,[7] in several species of animals including the dog, cat, hog and goat. In the operation the bile-duct and the pancreatic duct were implanted into the jejunum. Long-continued observations made on the dog, cat and hog following duodenectomy did not reveal any noticeable changes. The authors concluded that the function of the duodenum is not very different from that of the rest of the small intestine.

Intestinal Juice

No recent study of intestinal juice from farm animals appears to have been made. The older studies of Lehmann,[8] on a goat, and Pregl,[9] on a lamb, may be mentioned. Both of these workers used the fistular method of investigation.

Biliary System and Secretion

The hepatic ducts, the gall-bladder and its cystic duct, and the bile-duct constitute the extrahepatic biliary system. There are marked variations in this system in different species, as the study of Mann, Brimhall and Foster[10] has shown. Also there

are variations in different species in the relation of the bile-duct to the pancreatic duct, as the same workers have shown.[11]

The pressure under which bile is secreted has been studied[12] in a number of species, some possessing a gall-bladder and some not, and it was found that considerable variations occurred but that the presence or absence of a gall-bladder was not one of the causes. The secretion of bile is continuous, although the rate is variable. Its entrance into the bowel of animals possessing a gall-bladder is intermittent; in animals not possessing a gall-bladder, continuous.[13]

Reaction in Intestine

Graham and Emery[14] found that the reaction in the duodenum of the dog varied from pH 6.2 to 6.5, regardless of whether the dogs were fed a normal diet or a diet in which protein, fat or carbohydrate predominated. These findings are in harmony with the view of McClendon and co-workers,[15] who believe that the reaction in the intestine is determined more by the relative length of the bowel than by the character of the food. Grayzel and Miller[16] likewise found that high fat, protein or carbohydrate diet caused no notable change in the intestinal reaction in dogs. Slight acidity prevailed throughout the tract.

Long and Fenger[17] made a study of the reaction of ingesta obtained from the small intestine of hogs, yearling calves and yearling lambs slaughtered for food, and found that the reaction was more often acid than alkaline and that there was no definite variation in the different parts (upper, middle, lower) of the small intestine. The highest pH obtained was 8.05; the lowest, 6.39.

In a study involving the small intestine and cecal contents of a large number of cattle and horses slaughtered for food, Danninger, Pfranger and Schultes[18] obtained the pH values shown in table I.

Table I—*Reaction in intestine (pH)*

	Duodenum	Jejunum	Ileum	Cecum
Cattle	6.68	8.42	8.21	8.22
Horses	6.72		7.09	8.12

Intestinal Movements

Intestinal movements have been studied hardly at all in solipeds, ruminants and the pig; whereas, in the dog, cat, rabbit

and guinea pig they have been extensively studied. Alvarez's book, with a full bibliography, is the most important recent contribution to our knowledge of the mechanics of the digestive tract.[19] Since the animal usually used by Alvarez in his studies is the herbivorous rabbit, they should be of especial interest to veterinary physiologists.

According to Alvarez, food probably goes down the intestine, not because of the operation of the so-called "law of the intestine," as formulated by Bayliss and Starling, but because of graded differences in the muscular activity of different parts of the bowel. The upper part is most active, the lower part is least active; the intervening parts are of graded intermediate activity. In support of the gradient idea of peristalsis, Alvarez has shown the existence in the intestine of many, probably interrelated, gradients, *e. g.*, of rhythmicity, irritability, latent period, metabolic rate, catalase content.

Rate of Passage of Ingesta

Using as an indicator Sudan III fed to animals, Fish[20] determined the minimum interval of time necessary for the appearance of the dye in the feces, with the following results: cow 16 to 17 hours, goat 14 to 17 hours, man 15 to 25 hours.

Ewing and Smith[21] state that feces markers (rubber discs) and color indicators are not practicable means of determining the rate of passage of food residues through steers. By a method based on a digestion trial and a subsequent slaughter test, they found that the average time probably varies between 72 and 84 hours.

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Value of Cottonseed Products Discussed

Stockmen who need a feed high in protein to supplement home-grown roughages will find cottonseed meal or cake satisfactory, according to information given in Farmers' Bulletin 1179-F, "Feeding Cottonseed Products to Livestock," just issued in revised form by the U. S. Department of Agriculture. Cottonseed meal stimulates the appetite of fattening animals and causes them to consume more feed and likewise to make greater gains.

Cottonseed meal is a valuable protein feed for dairy cows, since, as the bulletin shows, one pound of good-quality cottonseed meal furnishes as much digestible protein as three pounds of wheat bran. The addition of cottonseed meal to rations for high-producing dairy cows, however, renders the rations satisfactory only as far as the protein requirement is concerned.

Beef cattle on pasture may be fed cottonseed cake or meal as a fattening ration with satisfactory results. Hogs also may be fed cottonseed meal in limited quantities as a protein supplement. Horses, however, should receive only cottonseed meal of good quality and in limited quantities, not more than one pound daily for a horse weighing 1,000 pounds, if digestive disturbances are to be avoided.

The bulletin shows how to compare various grades of cottonseed meal or cake and how to select the feed which supplies a pound of protein at the least cost. A number of suggested rations which include cottonseed products are listed and the results of several experiments show the value of supplemental feeding of animals on pastures. Copies of the bulletin may be obtained on application to the Office of Information, U. S. Department of Agriculture, Washington, D. C.

THE DRUMMOND PIPETTING MACHINE*

By M. F. BARNES, *Harrisburg, Pa.*

Division of Laboratories, Pennsylvania Bureau of Animal Industry

In our serum-test work, chiefly for Bang disease of cattle and pullorum disease of chickens, it is necessary to do considerable pipetting and we have experimented with various types of auto-

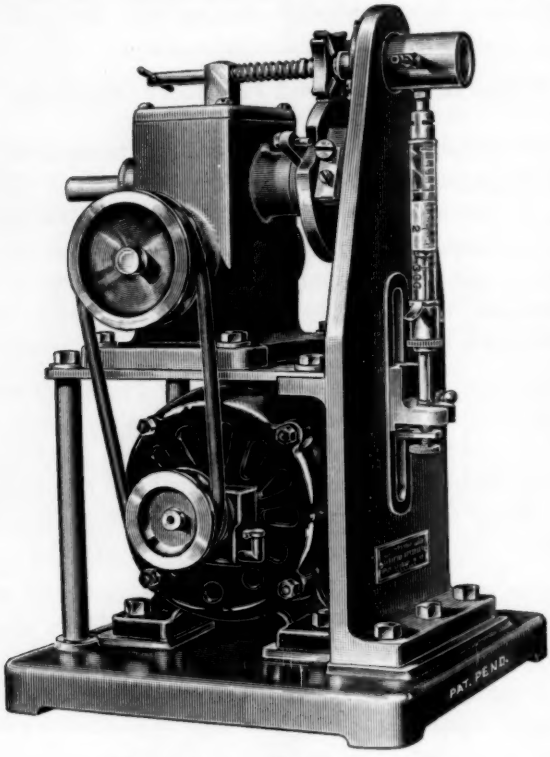

FIG. 1. Drummond pipetting machine

matic machines, electrically driven. It has been difficult to find a satisfactory machine. Under the strain of continued use, the machines which have been tried have developed leaky valves, which resulted in inaccuracy and unreliability. This condition made it necessary for use to search for a machine which would stand up under continued use, and which could be depended

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upon at all times. We believe we have found it in the one designed for us by Mr. L. E. Drummond, of Edw. P. Dolbey & Company, Philadelphia. We have given it a thorough test and find it comes up to all our requirements.

In the Drummond machine, the valve mechanism is constructed on an entirely different principle, being a simple four-way stopcock which is actuated at each end of the piston-stroke by a four-point-star wheel, similar in action to a motion-picture escapement. The plug of the stopcock is of stainless steel, ground into a shell of bronze, insuring a long term of use without repair or special attention. To clean this machine, it is necessary only to lift out the syringe and remove stopcock plug; the complete cleaning requires but a few moments.

The syringe is mounted vertically, tip up, and remains stationary. This makes it self-priming and precludes the possibility of air collecting therein.

The stroke of the piston may be adjusted to deliver from 0.1 to 2.5 cc with the 3-cc syringe, and up to 4 cc with the 5-cc syringe. Once set for the proper amount, the machine will deliver precisely the same volume at the rate of approximately 60 charges per minute. The regular stock B-D Sanalok syringe is used and may be inserted just as purchased, without alteration. Either a 3-cc or 5-cc syringe may be used.

The machine is sturdily built and takes up a space approximately eight inches square. Aside from a sound similar to the ticking of a clock, the machine runs almost silently.

Tuberculous Cattle Now Below Million Mark

Since initiating the cooperative campaign to eradicate bovine tuberculosis in 1917, state and federal agencies have located and slaughtered more than two-thirds of all the infected cattle in the country, according to Dr. Elmer Lash, of the U. S. Bureau of Animal Industry, who addressed the Southwestern States Tuberculosis Conference at Jackson, Miss., on January 22. During this period veterinarians engaged in this work tested and retested more than 75,000,000 cattle. Approximately 2,000,000 cattle were found infected with the disease, and all of these have been slaughtered. The consistent efforts of federal, state and county veterinarians have reduced the infection in cattle from 4 per cent, in 1922, to 1.7 per cent, on January 1, 1931. As a result there are now less than 1,000,000 tuberculous cattle in the United States.

SURGERY OF THE EYE (CANINE)*

By IRVIN F. BRENNING, *Syracuse, New York*

I have taken up three operations which I find common in my practice. First, the removal of the membrana nictitans or third eyelid. In nearly all cases where this is necessary, the glands on the inner surface are enlarged to such an extent that the entire membrane, with the enlarged gland, protrudes between the lids, even when the lids are closed. This enlargement is usually due to a tumorous growth of its gland tissue, the result of a chronic infection.

Treatment, other than surgery, is rarely, if ever, successful in reducing the size of the growth or destroying the infection. My method of removal, in any breed except hunting dogs, is to anesthetize the part to be removed by the use of a 2 per cent solution of butyn, if the dog be tractable. Otherwise, a general anesthetic must be used. While an assistant holds the dog's head and draws the lower lid downward, the edge of the membrane over the tumorous swelling is grasped with a pair of mouse-toothed forceps, drawn upward and snipped off with a pair of serrated scissors. Care must be taken not to injure the eyelids.

Any hemorrhage which may occur is easily controlled by the use of pressure over the lids with clean cotton packs. The post-operative treatment consists of a mild eye-wash and the use of a plain conjunctivitis ointment for a few days.

In hunting dogs I do not remove the entire membrane, but merely dissect the tumorous growth from the back of the lid.

ENTROPION

The second operation I wish to discuss is that which is done to relieve entropion. I find that this condition rarely responds to treatment other than surgical. It usually occurs in Chows and

*Presented at the twenty-first annual meeting of the Central New York Veterinary Medical Association, Syracuse, N. Y., November 20, 1930.

English Bulls, although I have seen it in other breeds and not long ago I did a successful operation on a cat.

The rolled-in part of the lid is usually more pronounced from the center to the outer canthus. To relieve this the patient must be given a general anesthetic, preferably ether. The skin below the eye is shaved and thoroughly cleansed. A crescent-shaped area is outlined with a scalpel, having the upper border about one-fourth inch below and parallel to the lid, with the outer point well beyond the canthus. Care must be taken in judging the area of the skin to be removed so that the desired result may be obtained. It has been my experience that a crescent one to one and one-half inches long and half an inch at the widest point will relieve a pronounced case. The skin within the outlined crescent is carefully removed and the edges of the wound drawn together with interrupted horse-hair sultures, starting in the center and working toward the ends.

The eye is immediately relieved. The stitches should be kept well greased to allay any itching. I find butysin picrate ointment (Abbott) excellent for this purpose. On the sixth or seventh day, the stitches may be removed.

Enucleation

The third operation is the complete enucleation of the eyeball. There are several conditions which call for this operation: First, traumatism is the most common and usually calls for immediate surgery. Second, infection, resulting either from the neglect of minor injuries or developing in spite of treatment. This is always very painful, especially after the infection has worked inside the eyeball. Third, unsightly blindness, which is usually accompanied by a purulent or watery discharge.

To perform the operation the patient is placed on the table in prone position and a general anesthetic administered by an assistant. With a sharp scalpel I cut through the skin of each eyelid parallel to and about three-sixteenths of an inch from the edge. This incision must extend completely around the eye. Then, while the assistant stands ready to keep the field of operation as free from blood as possible, the loosened edge is grasped with a pair of forceps and dissected down to the conjunctiva, which is then peeled from the underlying tissue down to the eyeball. All muscles are carefully dissected from the eyeball so as to leave as much tissue as possible in the cavity. This helps to fill in the cavity after the operation is complete.

The optic artery may be ligated after the eyeball is loosened, but it is scarcely necessary, as any hemorrhage which may occur after the artery is cut can easily be controlled.

After the eyeball has been removed, I press a pack of sterile gauze into the cavity and hold it firmly for a few moments. This pack is left until the opening is about closed. Interrupted sutures of horse hair are used, beginning at the inner canthus and placed close enough together to prevent any escape of blood if hemorrhage should continue after the removal of the pack.

The patient must be watched closely until completely recovered from the effects of the anesthetic, so that the wound will not be torn open.

If the pressure back of the stitches is very great at the end of the forty-eight hours, one or two of the stitches may be removed to permit the escape of the serum present. The clot or solid part is left, which, in case of no infection, organizes and forms scar tissue to help fill the cavity. The wound is then allowed to drain until healing is complete, which usually requires about eight or ten days.

In case of a blind or abscessed eye, however, where infection must be contended with, it might be necessary to irrigate the cavity daily with a mild antiseptic solution. I have never had a case which did not heal completely within fourteen days, although the appearance of the cases which were infected was never quite so good as those free from infection. More tissue had been destroyed and consequently a pit was left after healing.

A METHOD FOR THE COLLECTION OF BOAR SEMEN

By FRED F. MCKENZIE, *Columbia, Mo.*

Department of Animal Husbandry, College of Agriculture, University of Missouri, the United States Department of Agriculture co-operating

To facilitate a close examination of breeding boars the method here described was devised. By this method it is possible (1) to observe and study the copulatory act, (2) to examine the penis, and (3) to collect for examination or artificial insemination all the semen that may be discharged. For these purposes this scheme has proved simple and satisfactory. The idea is passed on in the

belief that the method can probably be used to advantage by others, including clinicians and veterinarians in the laboratory or in the field.

A photograph of the apparatus used is submitted. A soft rubber tube, 16 inches long, one end of which is fitted over the mouth of a test-tube, the other end over a $1\frac{5}{8}$-inch key-ring, and a clamp make up the outfit. The rubber is band tubing, $1\frac{3}{16}$ inches inside

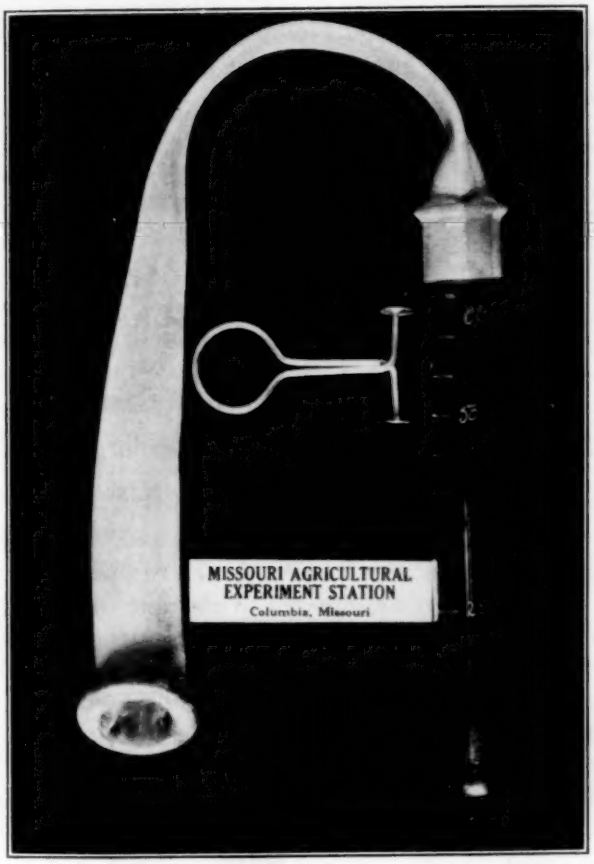

FIG. 1. Apparatus for the collection of boar semen.

diameter and $1\frac{1}{4}$ inches outside diameter, from the Arthur H. Thomas Company, Philadelphia. The test-tube was graduated by the writer. When about ready to make the collection the inside of the rubber tube is moistened, the funnel end (ring end) clamped and the whole apparatus immersed in warm water. This warming is necessary only in cold weather. A sow is tied to a wall

by a rope in her upper jaw; it is not necessary that she be in heat. The boar is admitted. When he mounts and attempts to copulate, the funnel end of the rubber tube (clamp removed) is placed near his sheath so he can pass the penis into the tube. The tube is now manipulated with a pulsating motion by the attendant's hand. This encourages continued copulation and ejaculation. The discharged semen runs down the rubber tube and is collected in the test-tube. Care must be taken not to press too vigorously against the sheath, else part of the contents of the preputial diverticulum will be forced into the semen-collector. Rubber gloves are worn.

As much as 72 cubic centimeters of semen have been collected at one time from an eight-month-old boar. The operation requires from 5 to 20 minutes, depending on the condition and disposition of the boar used.

THE INCIDENCE OF CANINE SURRA IN THE PHILIPPINES

A Report of Two Natural Cases

By LOPE M. YUTUC, *College of Veterinary Science, University of the Philippines, Los Banos, P. I.*

While surra is quite a common disease of Equidae in the Philippine Islands, yet its incidence in the canine family appears to be a clinical entity rare enough to warrant the description of the two natural cases here discussed. From the literature consulted, Musgrave and Clegg[1] reported this disease in the native dogs for the first time, in 1903. Smith and Kinyoun[2] reported it in horses, in 1901, and Curry,[3] in carabaos and cattle, in 1902. Mitzmain,[4] in 1913, conclusively demonstrated that *Tabanus striatus* is the main factor in disseminating the disease in horses in the Islands, which fact was confirmed by Kelser,[5] in 1927.

Laveran and Mesnil[6] reviewed its incidence in dogs as follows:

> Lingard records in various reports the occurrence of epizoötics of surra among sporting dogs introduced from England. He observed outbreaks in the island of Bombay, and in other parts of Bombay Presidency. He quotes the opinion of Evans that surra occurs among the dogs in the district of Karnal, in the Punjab.
>
> In 1891 a disease which appears to have been allied to surra was prevalent among several packs of hounds in Bombay. In 1893 Lingard saw an outbreak of surra among English foxhounds. Mention of spontaneous cases of surra has also been reported in Indo-China. During the epidemic in Mauritius a certain number of dogs died of surra.

Again in India, Allen,[7] in 1925, described this condition in a pack of foxhounds, complicated with distemper. In 1928, Sani[8] also observed it in an imported dog from Somaliland, in Milan. In this present paper it is intended to describe two natural cases of surra in dogs met with during the year 1929-1930, in the college clinic. Previous to this writing, it can be assumed with a certain degree of safety that there must have been cases of this malady, but they have been either not described or properly diagnosed thus justifying the recording of the condition here discussed.

Case Report 1

Subject: A male dog, about two years old, black, native, weighed eight kilograms and was used for hunting. The animal was admitted in the clinic July 5, 1929, with the history that he had been depressed for about a week and had a poor appetite. That morning, the owner claimed, the dog ate a little, which he vomited shortly afterward.

Symptoms: The animal could not maintain a standing posture, even when made to do so. Mucous membranes pale, pulse 124, respiration 66, and temperature 39.3° C. at the time of examination. The dog was greatly depressed in a semi-comatose condition. A slight edematous swelling was observed in the region of the hock; offensive odor from the mouth and dried exudate around the nostrils. Examination of the blood revealed numerous trypanosomes swimming in the cover-slip preparation of fresh blood. In the course of the disease, it was observed that the appetite was variable and entirely absent at times, resulting in emaciation and cachexia. Five days after its admission, icterus developed. Paroxysmal fever, coincident with the appearance of the organisms in the peripheral circulation, was noted. Weakness of the hind quarters was manifested by a swaying gait of the animal. Slight keratitis also developed in the course of the disease.

Diagnosis: Surra.

Treatment: Antimony and potassium tartrate was given intravenously, in doses increasing 10 mgs. at each injection, the initial dose being 10 mgs. The drug was dissolved in enough sterile physiological salt solution to make a 1 per cent solution. Four doses were injected at intervals of one day, the last dose being 40 mgs. To regulate the bowel movements, an occasional dose of magnesium sulfate was administered. For the poor appetite of the animal I. Q. S. tablets were added to the daily

medication. Blood examination was negative for a period of thirty-four days, up to August 7, 1929. However, the following day the organisms were observed in the blood. Again the dog received the tartar emetic treatment. It was negative up to September 4, 1929, when a second relapse occurred. Another course of tartar emetic alternately with sodium fluorid was tried with minute doses of the former injected epidurally, calculated according to the formula of Edwards,[9] in intrathecal injection in horses. On September 22, the animal was found dead after a lingering illness of 79 days.

For unknown reasons the animal was not posted.

Case Report 2

Subject: This case came from a locality about 30 kilometers from the College. The animal was a male, native dog, about three years old, brindle, and weighed about 20 kilograms. It was used mainly as a house guard, although the dog was taken along occasionally to the farm of the owner. It was admitted in the clinic, August 2, 1930. For several weeks the animal had shown a loss of appetite and a progressive cloudiness of the eyes, resulting in an impairment of sight. Previous treatment consisted of ophthalmic ointment and small doses of fluidextract of digitalis.

Symptoms: On clinical examination the mucous membrane was markedly icteric, with general depression of the animal. It seemed as though the animal wanted to sleep all the time. While the dog was under my care, it was observed at times in a semi-comatose attitude, oblivious of its surrounding. The animal was completely blind, due to a severe parenchymatous keratitis, the right eye being complicated with hydrophthalmus. The cornea also was yellow. Appetite was variable and entirely lost during the later course of the disease; hind quarters decidedly weak. Intermittent fever was similarly noted; animal greatly emaciated. Blood examination revealed trypanosomes.

Diagnosis: Surra.

Treatment: Sodium antimony tartrate was given intravenously in a proportionately larger dose, as it has been claimed to be less toxic than the potassium salt. Sixty-five mgs. dissolved in 4 cc of distilled water was injected every other day until six doses were given. Artificial Carlsbad salt also was administered to stimulate rapid elimination of the bile to the duodenum. To improve the appetite nux vomica was mixed with the milk given

to the animal. Blood examination remained negative until the animal died, August 21, 1930.

Autopsy: Postmortem examination was made and the following lesions were observed: Mucous membrane pale and petechiated. Blood was watery and abdominal fluid purulent. Liver weighed one kilogram, and fatty degeneration observed. The kidneys were pale in the pelvis, injected on the medulla and pale on the cortex. Cut surfaces moist and incision margins did not approximate; capsule easily peeled off. The stomach content was fluid, mucoid in character and white; the mucous membrane injected. The intestines showed a similar picture. Petechiae were noted. The spleen weighed 250 gms., was greatly increased in size, and bluish brown in color. The bladder contained about 10 cc of orange-colored urine and the mucous membrane was hemorrhagic. The lungs weighed 500 gms., were pinkish blue and soft; hepatized areas were observed; cut surface moist and small bits of lung tissue sunk in water. The heart contained clotted blood in both ventricles. The brain was congested.

Morphology and Behavior of the Trypanosome

The study of the behavior and morphology of the parasite was carried on in fresh blood and in stained preparations. In the fresh-blood, cover-slip preparations the protozoa have an eel-like motion. The motility varied in the specimens examined. The organism was observed tagging red blood cells in some of the slides. Blood smears were prepared, dried, fixed and stained either with Giemsa or Wright's stain. Observations in size, shape and other minute structures of the trypanosome were made from the stained slides prepared from both cases as well as from the inoculated animals. The organism measured from 20 to 30 μ in length, including the flagellum and from 1 to 2 μ in breadth, having a whipworm-like shape. The posterior end terminated in a somewhat blunt point, while the anterior narrowed to a sharp point. The undulating membrane was well defined with two or three folds, and began at or near the centrosome in the posterior portion of the parasite and ended anteriorly in a long flagellum. The nucleus was well marked and the protoplasm granular.

Experimental Inoculations

Immediately after the animal was secured in a recumbent position, 5 cc of blood was drawn aseptically from the saphenous vein. The volume of blood obtained was increased to 10 cc by

aspirating 5 cc of sterile physiological salt solution. Two white rats, one rabbit, one dog and one horse were inoculated simultaneously, each receiving 2 cc of the surra material. The same technic was followed in case 2. The results of the experiments are shown in tables I and II.

TABLE I—*Results of experimental inoculation in case 1*

DATE OF INOCULATION	SPECIES OF ANIMAL USED AND DESIGNATION	RESULT OF MICROSCOPIC EXAMINATION	DATE OF APPEARANCE OF ORGANISMS IN THE PERIPHERAL CIRCULATION	DATE ANIMAL DIED OR WAS KILLED
7-5-29	W. Rat 1	Positive	7-11-29	7-14-29
	W. Rat 2	Positive	7-11-29	7-14-29
	Rabbit 1			8-3-29
	Dog 1	Positive	7-17-29	8-1-29
	Horse 1	Positive	7-11-29	7-30-29

TABLE II—*Results of experimental inoculation in case 2*

DATE OF INOCULATION	SPECIES OF ANIMAL USED AND DESIGNATION	RESULT OF MICROSCOPIC EXAMINATION	DATE OF APPEARANCE OF ORGANISMS IN THE PERIPHERAL CIRCULATION	DATE ANIMAL DIED OR WAS KILLED
8-2-30	W. Rat 3	Positive	8- 6-30	8-10-30
	W. Rat 4	Positive	8- 6-30	8-10-30
	W. Rat 5	Positive	8- 7-30	8-10-30
	G. Pig 1	Positive	8-14-30	10-1-30
	G. Pig 2	Positive	8-14-30	10-6-30
	Dog 2	Positive	8-14-30	8-30-30

SUMMARY AND CONCLUSION

From the foregoing the following may be stated:

1. Two natural cases of surra in dogs are here reported.

2. The principal symptoms are: anorexia, keratitis and icteric condition of the visible mucous membranes, as well as the non-pigmented portion of the skin; paroxysmal fever and a decided weakness of the hind legs observed. Emaciation is a constant symptom.

3. From the morphological studies made and with the results obtained from experimental inoculations the protozoon described in this paper is *Trypanosoma evansi* Steele (1885), dog strain.

4. Tartar emetic, both the potassium and the sodium salts, failed to effect a cure in the two cases discussed.

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CHRONIC DIAPHRAGMATIC HERNIA

By R. W. JONES, *Athens, Ga.*

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Georgia State College of Agriculture

That an animal may live and be fairly serviceable for a considerable period of time, while suffering from diaphragmatic hernia, was evidenced recently by a case which came as a dissecting subject into the anatomy laboratory of the Division of Veterinary Medicine of the Georgia State College of Agriculture.

The animal was a small mule, weighing in the neighborhood of six hundred pounds and about fourteen hands high. No history was obtainable since he was purchased from a "dinky trader" who knew nothing of his past, and his condition was not suspected, before the postmortem examination, since he showed no incapacity to get about in the normal way.

Upon opening the thoracic cavity on the left side, it was discovered that a very great portion of the large colon occupied the position normally occupied by the left lung. The lung was contracted into a somewhat cylindrical mass, about five inches in diameter, in the upper part of the cavity. The structure of it seemed to be fairly normal except that the air-spaces were closed to a great extent from pressure.

The intestine had found its way into the thoracic cavity through a rent, about five inches in length, at the lower central part of the diaphragm just above the xiphoid cartilage of the sternum. The posterior mediastinum, ventral to the esophagus, had been destroyed and was entirely missing, and a part of the gut occupied this position between the heart and the diaphragm; a small part extended over into the right pleural sac.

The edges of the opening in the diaphragm were smoothly healed, indicating that the accident had occurred long since. There were slight adhesions between it and the apex of the cecum, which protruded only slightly through the wound. All the evidence led one to believe that this mule had lived and worked, probably for years, despite the displacement of the viscus and its permanent occupancy of the thoracic cavity.

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Rat Proofing Buildings and Premises. James Silver, W. E. Crouch and M. C. Betts. (Farmers' Bul. 1638. U. S. Dept. Agr., Washington, D. C., December, 1930.) pp. 25.

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The Significance of "Shift to the Left" in Differential Leucocyte Counts and the Nuclear Index as a Means for Interpreting and Recording. I. The Nuclear Index of Normal Blood and the Influence of Age. II. The Nuclear Index in Disease. Jules H. Gerard and Fred. Boerner. Reprint from *Jour. Lab. & Clin. Med.*, xvi (1930), 3, p. 300.

The "Shift to the Left" and Nuclear Index of Polymorphonuclear Leucocytes. John A. Kolmer. Reprint from *Jour. Lab. & Clin. Med.*, xvi (1930), 3, p. 327.

Effect of Pullorum Disease on Second Year Egg Production. Jacob Biely. Reprint from *Sci. Agr.*, xi (1930), 4, pp. 221-227.

Capillariasis Vesico-Urethralis of Silver Foxes. Fritz Volkmar. Reprint from *Vet. Med.*, xxv (1930), 12, pp. 3.

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Black Disease (Infectious Necrotic Hepatitis) of Sheep in Australia. A. W. Turner. (Bul. 46. Council for Scientific and Industrial Research, Melbourne, Australia, 1930.) Illustrated. pp. 141.

Red-Squill Powder in Rat Control. James Silver and J. C. Munch. (Leaflet 65. U. S. Dept. Agr., Washington, D. C., January, 1931.) Illustrated. pp. 8.

The U. S. Department of Agriculture is conducting experiments to reëstablish the musk ox in Alaska. A herd of thirty head has been imported from Greenland.

THE LESIONS IN EXPERIMENTAL AMEBIC DYSENTERY. Dale L. Martin. Arch. Path., x (1930), 4, p. 531.

Intestinal stasis, minute or gross, is an important factor in making it possible for *Endameba histolytica* to initiate its destructive attack. Examination of very early amebic lesions in the middle portion of the colon revealed that, outside the usual region of physiologic intestinal stasis, the cecum and the sigmoid, the lesions being at points of microscopic stasis, *i. e.*, the depths of grooves produced by normal mucosal folds. In the kitten, *Endameba histolytica* attacks first the most superficial portion of the intestinal mucosa, and destroys the epithelial cells of this region and the mucosal interstitial tissue with equal facility.

In the absence of intestinal obstruction, the organism does not invade the mucosal glandular crypts until their deeper expanded portions have become opened by a process of necrosis that has destroyed the superficial half of the mucosa. Tissue is destroyed by the action of a secretion produced by *Endameba histolytica*. Destruction of tissue is not produced by mechanical action. Except in few instances, the organism occupies only the necrotic zone resulting from the destruction of tissue. Conclusions are not drawn concerning the role of bacteria in the production or modification of amebic lesions, intestinal or hepatic, in the kitten.

THE LOCAL NEUTRALIZATION OF RABIES VIRUS. Claudio Fermi. Abst. Arch. Path., x (1930), 4, p. 622.

The dissemination of rabies virus subsequent to its subcutaneous injection can be prevented in rats if solutions of 1 to 10,000 mercuric chlorid, 1 to 1,000 silver nitrate, 2 per cent phenol, or 1 to 200 methylene blue (methylthionin chlorid U. S. P.) are injected immediately around the point of injection. After fifteen minutes, however, such injections are of no avail. The absorption of the virus through the nasal mucosa and rectum is so rapid that in fifteen minutes no method will prevent rabies.

Amputation of the tail, five hours after inoculation, was successful, as was also local passive hyperemia by means of elastic binders, which saved all of the animals tested, even when employed four hours after infection.

THE RESULTS OF ORAL ADMINISTRATION OF AMNIOTIN TO MONKEYS. J. A. Morrell, H. H. Powers, J. R. Varley and J. DeFrates. Endocrin., xiv (1930), 3, p. 174.

A total of 64 monkeys were given amniotin by stomach-tube. In 33 of those, when 400 or more units were given, menstruation resulted. In 2 of 4 monkeys, on 225 units, bleeding occurred. The remaining 27 on lower dosages did not respond. There was no difference in the amount necessary, whether the commercial preparation was used or a special glycerin-alcohol solution. A total of 23 monkeys were injected twice daily for 6 days. Eleven of these menstruated on dosages of 68 units or over. The remainder gave negative results. About 400 units are necessary when given by stomach-tube to produce menstruation. About 75 units when injected will do the same thing. So the ratio between the oral and subcutaneous dosage is about 5:1. Any greater amount than that required to produce menstruation has no added effect nor any undesirable results.

AN EPIDEMIC DISEASE OF THE DOMESTIC FOWL CAUSED BY A HITHERTO UNDESCRIBED ORGANISM OF THE SALMONELLA PULLORUM TYPE. Wayne N. Plastridge and Leo F. Rettger. Jour. Inf. Dis., xlvii (1930), 4, p. 334.

The authors describe an outbreak of a disease affecting adult barnyard fowls and young chicks. Although the causative agent attacks the same fermentable substances, and possesses essentially the same antigenic and agglutination properties as *Salmonella pjllorum*, it differs materially from the well-known pullorum disease organism in morphology, cultural requirements, and virulence for adult birds. Its virulence for adult birds is much greater than the virulence of ordinary strains of *S. pullorum*. Acute septicemia, marked pathologic changes and a mortality of 5 to 25 per cent occur. The organism fails to grow on ordinary nutrient agar plus 1 per cent glycerol. It develops abundantly on liver-infusion agar. Preliminary work on the serologic characteristics of this organism indicates a close serologic relationship between it and *S. pullorum*.

EXPERIMENTAL FIBROUS OSTEODYSTROPHY (OSTITIS FIBROSA) IN HYPERPARATHYROID DOGS. Henry Jaffe and Aaron Bodansky. Jour. Exp. Med., lii (1930), 5, p. 669.

The experiments show that parathyroid extract (parathormone Collip) can be injected into puppies in increasing amounts for long periods without fatal results. Thus time is allowed for bone changes to develop. Long continued injection leads to progressive decalcification and resorption of the existing bone, to fibrous replacement of the marrow, and to the production of the other features characteristic of ostitis fibrosa. Deformities eventually appear. It is safe to assume that the bone changes produced by hyperparathyroidization have the same pathogenesis as those observed in clinical cases believed to be instances of hyperparathyroidism, that is, cases with a negative mineral balance and decalcification of the skeleton.

EXPERIMENTAL MEASLES IN RABBITS. P. Belikoff, P. P. Dwijkoff and E. Truschina. Abst. Arch. Path., x (1930), 4, p. 622.

The authors injected rabbits with blood and filtrates of the pharyngeal mucosa from patients ill with measles, and in some instances fever, slight leukopenia and mucosal and skin reactions appeared. Similar results also were observed subsequent to the injection of a pure culture of a diplococcus recovered from patients with measles. Inflammation was found around the blood-vessels, with proliferation of the adventitial elements, frequently mixed with polymorphonuclear leucocytes. These changes were most marked in the skin; to a lesser degree in the larynx and trachea with, at times, hemorrhages in the mucosa. These changes were not observed following the injection of blood and mucosal filtrates from patients free from measles.

AMMONIUM MALATE AS A SOURCE OF NITROGEN FOR TUBERCLE BACILLI IN CULTURES. R. R. Henley and P. W. LeDuc. Amer. Rev. Tuber., xxii (1930), 5, p. 568.

The authors made an endeavor to find an inexpensive substitute for asparagine, which has generally been used as the chief source of nitrogen in synthetic culture media for the tubercle bacillus. A study was made of a variety of ammonium compounds. As substitutes for asparagine, urea and the ammonium salts of carbonic, malic, maleic, aspartic, citric, fumaric,

lactic, tartaric and succinic acids were used. The culture media containing urea, as well as those containing ammonium salts of carbonic acid and malic acid, afforded only very slight growth of bacteria. The medium containing the ammonium salts of aspartic, citric, fumaric, lactic, tartaric and succinic acids afforded fair amounts of growth. However, the ammonium salt of malic acid alone showed an efficiency approaching that of asparagine. Growth on synthetic media containing asparagine is nearly 2 gms. of bacteria (dry weight) per 100 cubic centimeters. Growth obtained by substituting ammonium malate for asparagine ranged from 1.5 to 1.9 gms. per 100 centimeters of culture fluid.

Differentiating Bovine and Human Tubercle Bacilli by Intracutaneous Injection in Rabbits. T. Toda. Abst. Arch. Path., x (1930), 5, p. 817.

Of the culture to be tested, 0.00001 mg. in 0.1 cubic centimeter is injected intracutaneously. A known bovine culture of high virulence should always be used as a control in each animal. Injecting one strain into the skin of one leg, three unknown cultures can be tested in one animal simultaneously. A caseous tuberculosis develops in the regional lymph-glands if a bovine culture is injected. This never occurs with human strains. Bovine strains make large persisting ulcers in the skin; human strains produce small healing primary infections.

A Study of Spirochetes in Chickens with Special Reference to Those of the Intestinal Tract. Minnie B. K. Harris. Amer. Jour. Hyg., xii (1930), 3, p. 537.

Three distinct types of spirochetes were harbored in the ceca of the normal adult chickens studied. They appear to be non-pathogenic and they have been designated tentatively as: (1) *Treponema caeci-gallorum*, (2) *Spironema caeci-gallorum* and (3) *Fusi-spirochaeta caeci-gallorum*. Young birds probably obtain these organisms by ingestion of food and water which has been contaminated by the feces of older birds. Once established in the caeca, the organisms tend to remain there throughout the life of the chicken. Whether they play a secondary role in infections has not been determined. Cultures from raw unfiltered material never yielded a satisfactory growth of spirochetes. *Treponema caeci-gallorum* is a small delicate organism from 5 to 7 microns long and in cultures up to 12 microns, 0.3 to 0.5 microns

in width and from 5 to 12 spirals. It is extremely motile. It passes through a Mandler filter as a spirochete and can be cultivated on a medium containing agar, ascitic fluid and sterile rabbit or guinea pig liver in test-tubes overlaid with sterile paraffin oil. *Spironema caeci-gallorum* is an active organism from 8 to 10 microns long, 0.5 to 0.75 microns in width, with pointed ends, and from 3 to 7 loosely coiled spirals. The organism is not filtrable and cannot be successfully cultivated. *Fusi-spirochaeta caeci-gallorum* is a strikingly distinct organism. It is a fusiform bacillus of various lengths from 5 to 15 microns, and from 1 to 2 microns wide at the center, which may take on a spirochetal motion. The organism was observed in raw material, not in the filtrate, but appeared and persisted in the cultures with the treponemas. Sections of the cecum stained according to the Levaditi method showed a penetration of the epithelial lining of the crypts by *Treponema caeci-gallorum*, while *Spironema caeci-gallorum* and *Fusi-spirochaeta caeci-gallorum* were present in the lumen of the crypts.

A Comparison of Factors Influencing the Agglutination Test for B. Abortus. B. S. Henry and J. Traum. Jour. Inf. Dis., xlvii (1930), 5, p. 367.

In the tube method of testing, formolized antigen has a tendency to intensify or cause proagglutination with human, bovine and porcine serums to such an extent that occasionally strongly positive serums might be missed in routine mass testing. In the same serums tested with phenolized or tricresolized antigen, the interference is absent or reduced to such a point that it is not misleading. Incubation over night at about 37° C. and then for 24 hours at room temperature gives better results than other methods tried. Variations in the opacity of the antigen within fairly wide limits have no great influence on the correct interpretation of results. A rapid and satisfactory method of reading tests based on the clearing of the fluid and manner of the distribution of the agglutinated and non-agglutinated bacteria at the bottom of the tube is given by the authors.

Boric Acid for the Preservation of Milk Naturally Infected with Brucella Abortus. J. Traum and B. S. Henry. Jour. Inf. Dis., xlvii (1930), 5, p. 380.

Since satisfactory icing or other means of refrigeration is frequently not practicable and milk samples may reach the

laboratory in a condition that causes the death of the animals into which they are injected, or samples may undergo sufficient souring to destroy *B. abortus*, the authors attempted to preserve milk samples by the use of boric acid. One per cent boric acid preserved the milk for at least 253 hours at ice-box or room temperatures and for 118 hours at 37° C. to the extent that milk was not fatal to guinea pigs and it did not destroy the Brucella organisms as indicated by infection induced in these animals.

Experiments in Dosage of Carbolized Antirabic Vaccine. J. Taylor and K. N. Menon. Indian Jour. Med. Res., xviii (1930), 2, p. 711.

This series of experiments was limited to 160 rabbits. A dosage of carbolized vaccine considerably in excess of the equivalent human dose of the standard treatment results in improvement of the protection obtained in the case of rabbits. The removal of brain substance from the vaccine by filtration greatly reduces its immunizing value. However, the preliminary extraction of ether-soluble material from the fixed-virus brain does not affect the value of the vaccine. The authors observed throughout the course of their experiments the considerable difference in the virulence of street viruses obtained even in one locality.

Serological Studies in Experimental Coccidiosis of Rabbits. George W. Bachman. Amer. Jour. Hyg., xii (1930), 3, p. 624.

Complement-fixation tests were made on 19 rabbits infected with *Eimeria stiedae* and *Eimeria perforans*. Six gave strongly positive reactions in serum dilutions of 128 to 256, eight gave weakly positive reactions in serum dilutions of 32 to 64. Two showed no increase above the non-specific reaction of the serum, while the titres of two rabbits fell below the initial control tests. In artificially immunized rabbits precipitins were detectable in high concentrations for five days after the last injection of the antigen, then gradually disappeared and were not demonstrable after the 45th to 50th days. In rabbits experimentally infected with *E. perforans* and *E. stiedae*, precipitation tests were uniformly negative. The results of 932 benzoin flocculation tests on 66 rabbits 5 weeks to 4 years old show that the flocculation of the sera of rabbits infected with *E. perforans* and *E. stiedae* is distinctly increased over the flocculation of normal rabbit serum.

There is apparently demonstrated in this test some specific response to coccidia. Measurement of the index of refractivity failed to demonstrate any difference between the sera of normal rabbits and of rabbits infected with the two species of coccidia. Seventy-four surface tension determinations were made on the sera of infected rabbits. These determinations seem to be of little value as a measure for differentiating the sera of normal and coccidia-infected rabbits.

Immunity in Experimental Coccidiosis of Rabbits. George W. Bachman. Amer. Jour. Hyg., xii (1930), 3, p. 641.

A study was made of 57 rabbits from a colony shown to be free of the common parasites with the exception of intestinal coccidia (*Eimeria perforans*). Rabbits from 4 months to 4 years of age that had lived in this colony and had previous infections of *E. perforans* were immune to experimental infection with the same organism. Rabbits of the same colony, immune to *E. perforans* infection, were not resistant to infection with *E. stiedae*, independent of age. The attempts at active and passive immunization failed to protect rabbits against infection with *E. perforans*.

Jaundice in Experimental Coccidiosis of Rabbits. George W. Bachman and Paris E. Menendez. Amer. Jour. Hyg., xii (1930), 3, p. 650.

Tests on 31 rabbits infected with *Eimeria stiedae* gave 111 positive reactions for bilirubin, of which 90.1 per cent were direct, 8.1 per cent indirect and 18 per cent were biphasic. One rabbit, slightly infected, failed to give a positive reaction. The large variations in the quantitative estimates of bilirubin contents in these rabbits are probably due in part to the fact that the determinations were made at intervals of days and not at intervals of hours and also to the amount of epithelial destruction of the bile-ducts during the various stages of schizogony. The intensity of the infection found in some of these rabbits could have caused the rupture of small blood-vessels and the destruction of liver cells, which would be sufficient to account for the small percentage of hemolytic jaundice. The high percentage of direct immediate reactions obtained in these tests indicates obstructive jaundice. Microscopic studies of stained sections of livers in the early stages of schizogony show the epithelial cells of biliary passages parasitized and swollen. In the

latter stages of the infection the biliary passages may be seen plugged with proliferated cells of the epithelium and oöcysts. In heavy infections the gall-ducts and gall-bladder are plugged with the numerous orange-yellow-colored oöcysts. The compaction of these oöcysts gives rise to obstructive jaundice.

HEXYLRESORCINOL IN EXPERIMENTAL TETANUS. George E. Colemen. Jour. Inf. Dis., xlvii (1930), 5, p. 410.

Hexylresorcinol S. T. 37, when mixed with tetanus toxin in 16.6 per cent solution, is capable of neutralizing 10 and less than 100 minimal lethal doses. It is also bactericidal for the vegetating forms of tetanus bacilli within half an hour *in vitro*, but while strongly sporicidal does not kill resistant spores in 12 hours. Hexylresorcinol S. T. 37, when injected into the devitalized or necrosed tissue of guinea pigs infected with tetanus spores, is without effect in modifying the course of the disease.

PRECIPITIN AND COMPLEMENT-FIXATION TESTS ON DOG SERA WITH ANTIGEN FROM THE DOG HOOKWORM, ANCYLOSTOMA CANINUM. John E. Sturnberg. Amer. Jour. Hyg., xii (1930), 3, p. 657.

Dried filariform larvae and adults of the dog hookworm, *Ancylostoma caninum*, extracted with acid and alkaline saline solutions, were found to be highly antigenic when injected intravenously into rabbits; alkaline saline extracts were preferable to acids. Slight differences between larval and adult antigens were found, mainly the production of a higher titre by the adult extracts, but the two could hardly be considered serologically distinct. The antibodies produced in immunized rabbits were species specific in dilutions of 1:400 or over, and group specific in dilutions of from 1:1000 to 1:4000. Non-specific reactions were obtained with antigens prepared from other intestinal helminths but never in dilutions above 1:1000 and usually in dilutions of only 1:100. No evidence could be found that the antigenic action was due to contaminating bacteria. The serum of dogs showed a non-specific complement-fixation titre with these antigens as high as 1:500, and this reaction was not destroyed by heating to 62° C. for 15 minutes. Dogs parenterally injected with larval and adult antigens failed to show any precipitins in the sera or any complement fixation in excess of the non-specific level. Dogs experimentally infested with hookworms, either by single infection or after several infections, failed to show antibodies in their sera up to seven weeks after infestation.

Regular Army

Captain Louis G. Weisman is relieved from duty at Ft. Benj. Harrison, Ind., and directed to proceed to New York City and sail on transport scheduled to leave that port on or about February 19, 1931, for the Panama Canal Department, for duty with the Medical Department.

Captain Joseph H. Dornblaser is assigned to duty at Ft. D. A. Russell, Texas, effective upon completion of his present tour of foreign service in the Panama Canal Department.

Orders assigning Major Christian W. Greenlee to duty at Ft. D. A. Russell, Texas, are amended so as to assign him to duty at Ft. Benj. Harrison, Ind., to take effect upon completion of his present tour of foreign service.

Veterinary Reserve Corps

Transfers

Mayo, Nelson Slater.....Lt. Col....transferred to Auxiliary Reserve, Nov. 17, 1930

New acceptances

Pedigo, Geo. Wm.....Capt.....Maple Driveway, Glasgow, Ky.
Lamoreaux, Florance Morris.1st Lt.....R. R. 1, Comstock Park, Mich.
Mimnaugh, John Joseph.....1st Lt.....348 Migeon Ave., Torrington, Conn.
Westerberg, Ralph Victor....1st Lt.....403 W. Main St., New Britain, Conn.
Brinkman, Norman Henry...2nd Lt.....Colo, Iowa
Moss, Lloyd Charles.....2nd Lt.....Board of Agri. & Forestry, Honolulu, T. H.
Parshall, Charles Jonas.....2nd Lt.....Vet. Div., Univ. Calif., Berkeley, Calif.
Smit, Walter.....2nd Lt.....Alton, Iowa

Promotions

King, Edwin Doc, Jr.....To Major.....406 N. Ashley St., Valdosta, Ga.

Notice of Examination for Appointment in the Veterinary Corps, Regular Army

Examination for the purpose of qualifying candidates for appointment in the Veterinary Corps, Regular Army, to fill existing vacancies, will be held within the continental limits of the United States, March 16 to 21, 1931, inclusive.

Application blanks, Form No. 62, A. G. O., may be obtained from The Adjutant General or The Surgeon General, U. S. Army, Washington, D. C., or from the Commanding Officer or Surgeon of any military post or station, and when completed should be forwarded direct to The Adjutant General, U. S. Army, Washington, D. C.

The applicant must be a male citizen of the United States between the ages of 21 and 29 9/12 years, and a graduate of a recognized veterinary college.

Congress of Comparative Pathology

The Second International Congress of Comparative Pathology will meet in Paris, France, October 14-18, 1931. Research workers and others interested who expect to attend this meeting, and who desire to participate in the proceedings, should at their earliest convenience address one of the members of the Committee named below:

Dr. George W. McCoy, Chairman, Director of the National Institute of Health, Washington, D. C.

Dr. Milton J. Rosenau, Professor of Preventive Medicine and Hygiene, Harvard Medical School, Boston, Mass.

Dr. M. B. Waite, Principal Pathologist, Office of Horticultural Crops and Diseases, Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.

Dr. L. R. Jones, Department of Plant Pathology, College of Agriculture, University of Wisconsin, Madison, Wis.

Dr. V. A. Moore, New York State Veterinary College, Cornell University, Ithaca, N. Y.

Dr. J. R. Mohler, Chief, Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.

Will Judy Broadcasts on Dogs

Chappel Brothers, Rockford, Ill., manufacturers of Ken-L-Ration dog food products are sponsoring a weekly series of radio dog talks over Station WGW (Chicago Tribune Station, Chicago) every Sunday afternoon at 2:30 o'clock, C.S.T. These are given by Will Judy, editor of *Dog World*, and should create considerable interest and favorable publicity for dogs in general. Tune in on any Sunday afternoon and you will enjoy Mr. Judy's talks.

Bureau Transfers

Dr. M. M. Woods (U. S. C. V. S. '18) from Fargo, N. Dak., to New York, N. Y., on meat inspection.

Dr. R. J. W. Briggs (Chi. '02) from Sioux Falls, S. Dak., to Norfolk, Nebr., on tuberculosis eradication.

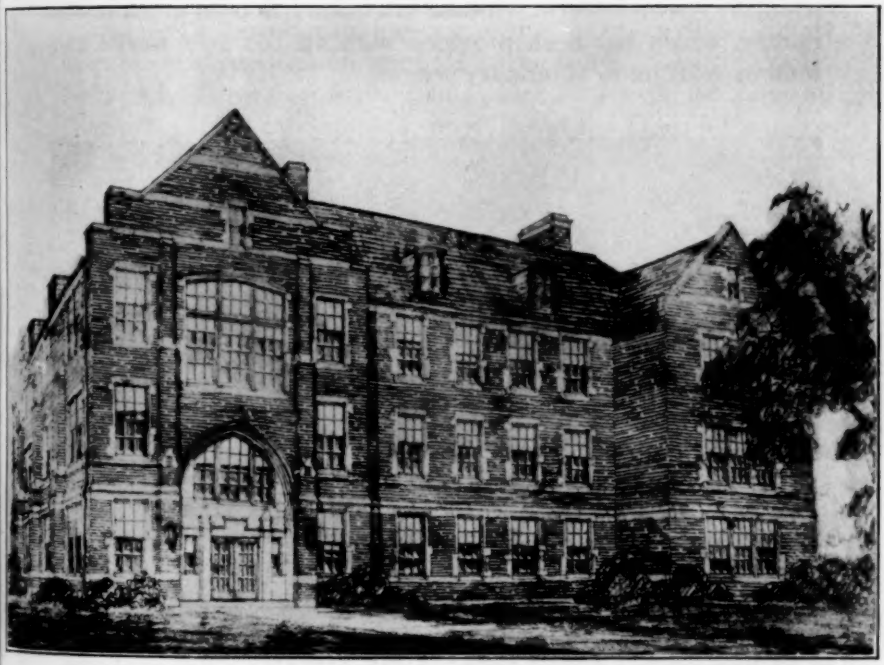
New Veterinary Building at East Lansing

As one indication of the faith which Michigan has in the future of veterinary medicine, one might point to the new building recently completed at Michigan State College, East Lansing, at a cost of approximately $100,000.00 exclusive of equipment. The new structure will house the departments of Anatomy and Animal Pathology, headed respectively by Drs. F. W. Chamberlain and E. T. Hallman, of the Veterinary Division of the College.

The latest addition to the veterinary unit at East Lansing occupies a prominent location on the campus—said to be one of the most beautiful in the world—near the Surgery and Clinic Building. It was planned in such a way that an addition may be made later, to house the Department of Bacteriology and Hygiene. The dimensions are approximately 100 feet front by 60 feet deep. The building is of fire-proof brick and concrete construction, three stories in height, with a spacious attic. Walls are of yellow tile; floors of offices, corridors and stairs are terrazo; all other floors are concrete, finished with thoroseal, a stain-proof floor dressing.

The first floor contains two rooms for gross anatomy, an autopsy room, mortuary and locker-rooms. The floors of the mortuary and large gross anatomy rooms are depressed about five feet below grade to provide head-room of about fourteen feet. Dr. Chamberlain has designed a very ingenious set of vats for embalming and storing cadavers of all domesticated and wild animals. A track conveys the larger animals from the operating-tables to the vats or to the dissecting-room. The small gross anatomy room is for osteology and dissection of detached parts. The autopsy room (Department of Animal Pathology) is modern in all respects. It has laboratory facilities, electric refrigerator and high-pressure steam. An incinerator will be provided.

The second floor is devoted exclusively to the Department of Anatomy. There is a well-lighted museum and a laboratory for the study of gross anatomy of the smaller animals. The laboratory for histology and embryology is commodious and well-lighted. Adjacent to it is a large preparation-room, which also communicates with the small laboratory for students in anatomical technic. This floor contains the only lecture-room in the building. Temporarily one of the offices will serve as a library.

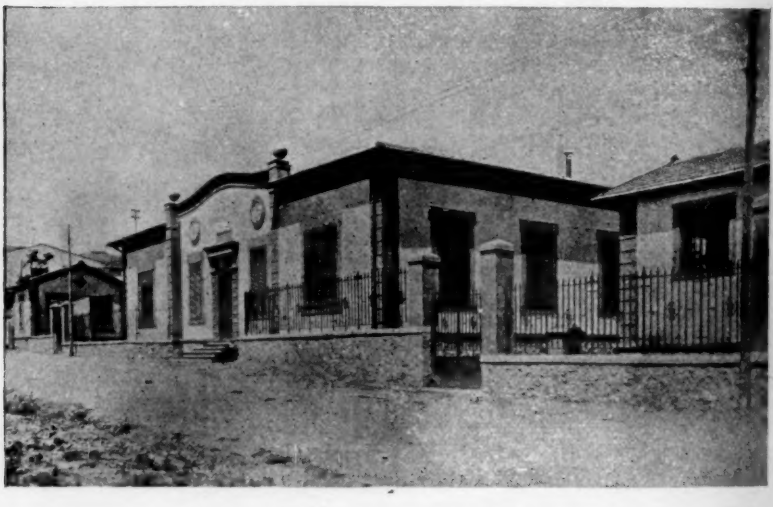


ANATOMY AND ANIMAL PATHOLOGY BUILDING, MICHIGAN STATE COLLEGE

The third floor is occupied by the Department of Animal Pathology and the pathologist attached to the State Department of Agriculture. There are seven private research laboratories, two offices, three student laboratories and a large pathological museum. High-pressure steam for autoclave operation is available on this floor and in the attic above. The latter is heated and lighted and is used for experiment animals and storage. It has a wash-room and sterilizing-room for glassware and an elevator connects this floor with the autopsy room.

Experiment Station in Sicily for Infectious Diseases of Animals

In Italy infectious diseases of animals are combatted by means of regional institutions (Stazioni Zooprofilattiche). The newest of these was opened recently in Palermo. The institution is composed of five buildings, the central one containing the offices and laboratories, the others being used for the housing of the animals under supervision. The equipment of the laboratories is the most modern obtainable and provides facilities for highly scientific research work. Special attention has been given to the library, which has been provided with all the best works and reviews relating to veterinary science.



EXPERIMENT STATION, PALERMO.

We think it of interest to give a résumé of the program of the work undertaken at the Station:

1. The diagnosis of infectious diseases by means of the examination of pathological specimens sent to the laboratory by veterinarians and others, and also by personal visits of the staff to the infected farms.
2. The preparation of vaccines and serums.
3. The study of the prevalent diseases in Sicily.
4. The holding of courses in theory and practice for veterinarians.
5. The conducting of intensive propaganda among the breeders of cattle in Sicily, so as to enable them to cooperate in the fight against disease.

The institution is presided over by the Prefect of Palermo, S. E. Albini, and is under the direction of Prof. Dr. Mirri.

NEBRASKA STATE VETERINARY MEDICAL ASSOCIATION

The thirty-third annual meeting of the Nebraska State Veterinary Medical Association was held at the Hotel Lincoln, Lincoln, Nebraska, December 9-10, 1930. Dr. F. Perrin, of Lincoln, presided. Mr. S. A. Sorenson, president of the Lincoln Chamber of Commerce, extended a most hearty and cordial welcome to the Nebraska veterinarians, and Dr. C. C. Hall, of Omaha, made an appropriate response.

The minutes of the previous annual meeting were read and approved and various committee reports received. Dr. C. H. Hays, chairman of the Resolutions Committee, presented timely and appropriate resolutions, copies of which were sent to the Nebraska Secretary of Agriculture. Also, a resolution was adopted opposing House Roll No. 7884, and a copy of the resolution was sent to each member representing Nebraska in the 71st Congress.

The first paper presented was entitled "Sodium Bicarbonate in Influenza Type of Diseases," by Dr. R. W. Hixon, of Falls City. This paper was full of many interesting experiences of the writer in connection with the use of this drug in the treatment of equine influenza, so-called shipping fever of cattle, and flu in hogs. He outlined several ways of administering sodium bicarbonate.

After lunch, Dr. E. A. Benbrook, of Iowa State College, presented "Animal Parasites." He dealt with the most common parasites, giving their life cycles, intermediate hosts, methods of attaching to the hosts, means of eradicating the parasites, as well as methods of prevention. Dr. Benbrook also dwelt on the financial importance of parasitic diseases to the live stock industry of the state. The paper was illustrated with lantern-slides, thus making the subject matter more clearly understood.

Dr. L. Van Es, of the University of Nebraska, presented a paper, "Metabolic Bone Diseases." This paper introduced a new field of thought. It was pointed out that these disorders were due to an improper food supply and could be prevented if the necessary elements were supplied, namely, calcium and phosphorus, of which the latter is the more important. It is most abundant in bone meal and cod-liver oil. Direct sunshine is also of great importance in connection with the condition described by Dr. Van Es.

Dr. A. T. Kinsley, of Kansas City, Mo., gave a talk on "The Significance of Pathology to the Veterinary Practitioner." This address took us back to our class-room days. Dr. Kinsley reviewed the various ways and means by which infection may be carried from one part of the body to another, the organs thus affected and the relation of one diseased organ to another. Dr. Kinsley also described the pathological conditions produced and their appearance on autopsy, outlining the difference between physiologic and pathologic changes. Dr. Kinsley also conducted a round-table discussion on swine diseases.

Dr. D. W. Hurst, of Tecumseh, gave a very interesting report of his trip to the Los Angeles convention of the A. V. M. A., the past summer. From this we would judge that the national association had a very successful and enjoyable meeting in the great weather state.

Dr. B. A. Beach, of the University of Wisconsin, gave an interesting talk on "Diseases of Poultry." He outlined the causes, methods of spread, treatment and prevention of the various diseases. Dr. Beach mentioned the importance of avian tuberculosis, as well as bacillary white diarrhea. He described the tests used for detecting the presence of both diseases. Dr. Beach laid a great deal of emphasis on the different disorders due to improper methods of feeding and the lack of sanitary surroundings for poultry.

At the morning session of the second day, Dr. Beach resumed the subject of poultry diseases and pointed out that vitamin D is the most important of the vitamins in successful poultry-raising. He stated that it was most abundant in cod-liver oil. Rickets was referred to as a very common disease of growing chicks and the best method of combating it was by feeding cod-liver oil, administering calcium salts and providing exposure to direct sunlight. Osteomalacia was described as being very common among mature birds and this condition can readily be prevented by

feeding oyster shells. When vitamin A is lacking from the diet, eye disorders are likely to follow. Dr. Beach illustrated his talk with a number of lantern-slides.

At the afternoon session, Dr. C. P. Fitch, of the University of Minnesota, gave a very timely and interesting talk on "Infectious Abortion in Cattle," its method of spread, prevention and the application of the agglutination test, as well as results obtained from the test. Dr. Fitch also dwelt on some of the regulations which are now in effect governing the interstate movement of cattle effected with abortion. He also mentioned the possibility of human beings contracting undulant fever by consuming milk from cows affected with the disease. He also used lantern-slides in connection with his talk, thus adding greatly to the interest of the subject.

Dr. J. E. Weinman, of Lincoln, presented a paper on "Parturient Toxemia," which was followed by a very spirited discussion.

The election of officers for the ensuing year resulted as follows: President, Dr. D. E. Trump, Utica; vice-president, Dr. C. C. Hall, Omaha; secretary-treasurer, Dr. E. C. Jones, Grand Island; members of the Executive Board, Dr. J. E. Weinman, Lincoln, and Dr. L. E. Hines, Spencer. Ten veterinarians were elected to membership.

The social side of the meeting was marked by a very delightful banquet, Tuesday evening, attended by about 200 members and guests. Vocal and instrumental music was provided. Chancellor E. A. Burnett, of the University of Nebraska, was the after-dinner speaker. The remainder of the evening was spent dancing and visiting, and everybody appeared to have a good time.

B. WITT, *Secretary-Treasurer.*

CUMBERLAND VALLEY VETERINARY CLUB

The Cumberland Valley Veterinary Club met at the Molly Pitcher Hotel, Carlisle, Pa., the evening of December 11, 1930, with about sixty-five veterinarians and their wives in attendance. A turkey dinner was the first number on the program. Then Dr. C. J. Marshall, of the University of Pennsylvania, spoke on the European trip in connection with the International Veterinary Congress. He was followed by Dr. E. L. Stubbs, also of the University of Pennsylvania, who reported his observations on the Fourth World's Poultry Congress.

B. SCOTT FRITZ, *Reporter.*

STATE VETERINARY MEDICAL ASSOCIATION OF TEXAS

Seventy-five members of the State Veterinary Medical Association of Texas convened at Wichita Falls, January 12-13, 1931, for the eighth semi-annual meeting of the Association. Quite a number of veterinarians from Oklahoma were in attendance, as well as representatives from several other states.

The meeting was called to order Monday morning, promptly at 8:30, by the President, Dr. T. O. Booth, of Temple. Dr. H. T. Wood, pastor of the First Christian Church, delivered the invocation, after which Mayor Walter Nelson, Jr., welcomed the veterinarians to Wichita Falls.

A response to the address of welcome was made by Dr. H. L. Darby, of Fort Worth, who complimented Wichita Falls and the local Health Department, citing work now being done by the city toward the insurance of healthfulness in the meat and milk supply, as well as the efforts of dairymen, in the vicinity of Wichita Falls, to limit their herds to disease-free animals. "We veterinarians," Dr. Darby said, "are proud to belong to a profession that is of benefit to all humanity, for by keeping animals and live stock healthy, we are protecting human lives."

Monday afternoon, Dr. F. B. Green, of the State Department of Health, at Austin, spoke on the milk control program in Texas. Dr. A. E. Wharton, of the Texas A. & M. College, College Station, presented the subject, "Side Lights of Municipal Milk and Meat Inspection." Dr. Ashe Lockhart, of Kansas City, Mo., spoke on abortion disease. Dr. T. O. Booth, of Temple, addressed the gathering on the problem presented by Bang abortion disease, especially as it confronts the veterinarian in private practice.

Dr. J. V. Lacroix, of Evanston, Ill., gave a very practical discussion on small-animal practice. Dr. C. E. Salsbery, of Kansas City, Mo., chose canine distemper as the subject of his address. Dr. P. W. Burns, of the Texas A. & M. College, College Station, covered "New Drugs of Importance to Veterinarians."

Tuesday morning, Dr. N. F. Williams, state veterinarian of Texas, opened the program with a discussion of the proposal for the affiliation of state and provincial veterinary associations with the American Veterinary Medical Association. Dr. H. V. Cardona, of Fort Worth, spoke on veterinary conditions in Mexico. Dr. R. P. Marsteller, of the Texas A. & M. College, College Sta-

tion, presented "Competent and Adequate Veterinary Service Essential to a Successful Live Stock Industry." Dr. T. O. Booth, of Temple, read a paper on a treatment for acute aseptic pododermatitis.

Members of the Ladies' Auxiliary were entertained at a theatre party, in the afternoon, following a luncheon at the Women's Forum, at 12:30.

The banquet was held Monday evening, with Dr. N. F. Williams officiating as toastmaster. A very enjoyable program of entertainment featured the banquet, including musical numbers, readings and dances. The after-dinner speakers included Mr. C. I. Francis, local attorney; Mr. Maurice Cheek, president of the Junior Chamber of Commerce of Wichita Falls; Dr. A. H. Douglass, head of the municipal Water Department; Dr. C. E. Salsbery and Dr. Charles D. Folse, of Kansas City; Dr. W. G. Gregory, of Fort Worth; Dr. T. T. Christian, of Waco; and Dr. J. V. Lacroix, of Evanston, Ill.

D. PEARCE, *Secretary-Treasurer.*

CHICAGO VETERINARY MEDICAL ASSOCIATION

At the regular monthly meeting of the Chicago Veterinary Medical Association, held at the Hotel Atlantic, Chicago, Ill., January 13, 1931, a large number of members and guests were entertained with motion-pictures showing the production of biologics. The pictures were shown by Mr. Stoner, of Parke, Davis & Company, Detroit. Mr. H. Cannon, of the Abbott Laboratories, gave an interesting talk on vitamins.

Visiting veterinarians are always welcome to attend meetings of the Association.

C. L. MILLER, *Secretary.*

SOUTHWESTERN TUBERCULOSIS CONFERENCE AND MISSISSIPPI VETERINARY MEDICAL ASSOCIATION

The 1931 Southwestern Tuberculosis Conference was held at the Edwards Hotel, Jackson, Mississippi, January 21-22, 1931, in connection with the regular annual meeting of the Mississippi Veterinary Medical Association. Hon. Walter Scott, mayor of Jackson, extended a hearty welcome to the Conference and Dr. E. Pegram Flower, state veterinarian of Louisiana, responded.

Dr. Henry Boswell, president of the Southwestern Tuberculosis Conference, complimented the veterinary profession for its progress in the control of tuberculosis of animals and for the achievements of its members in reducing the menace of bovine tuberculosis infection in humans, with special reference to such infection in children. He showed unusual knowledge of veterinary conditions and practices and a thorough appreciation of the public-health problems confronting both the medical and the veterinary profession. Dr. Boswell, who is superintendent of the Mississippi Tuberculosis Sanitarium, is president of the National Tuberculosis Association and widely recognized as an authority on tuberculosis.

Dr. Wm. Moore, state veterinarian of North Carolina, presented a paper on "The Economic Importance of Eradicating Tuberculosis," which reviewed the results of some careful analytical studies in this field. The discussion of his paper was ably led by Dr. N. F. Williams, state veterinarian of Texas.

In the absence of Dr. May F. Jones, of Laurel, Miss., her paper on "Methods of Tuberculosis Control in School Children," was read by Dr. W. E. Noblin, Hinds County Health Officer.

Dr. N. M. Parker, Director of Meat and Milk Inspection, Mississippi State Board of Health, presented a paper on "A Tuberculosis-Free Milk Supply," in which he embodied some of his interesting experiences in dairy sanitation. Dr. Henry Boswell gave a most enlightening discourse on "The Relationship of Bovine Tuberculosis in Humans."

Dr. J. R. Ricks, epidemiologist, Mississippi State Board of Health, gave a paper discussion on "Relationship of Medical Doctors to Veterinarians," in which he emphasized the interdependence of these two professions in public-health control.

Dr. Elmer Lash, Tuberculosis Eradication Division, B. A. I., Washington, D. C., gave a most instructive paper reviewing "Area Tuberculosis Eradication in the United States," and showing the remarkable progress made since 1917 in decreasing the incidence of tuberculosis in cattle and hogs as established by retesting and postmortem examinations at slaughter-centers under federal supervision.

Dr. J. H. Bux, state veterinarian of Arkansas, gave a report on "The Accredited Area Work in Arkansas," and explained the plan of bunching cattle as developed by tuberculosis workers in Arkansas, to facilitate the handling of large numbers of cattle in area work.

Prof. W. F. Bond, Mississippi Superintendent of Education, discussed public-health work as conducted in the public schools of Mississippi. Dr. F. F. Brown, retired veterinarian, formerly vice-president of the Kansas City Veterinary College, presented a most scholarly paper on "Rabies," which was followed by considerable discussion.

The Southwestern Tuberculosis Association accepted the invitation extended by Dr. Flower, state veterinarian of Louisiana, to meet in New Orleans in 1932. The meeting date and the presiding officers will be announced at a later date.

At a business meeting of the Mississippi Veterinary Medical Association, presided over by Dr. C. B. Cain, President, Hon. J. C. Holton, Commissioner of Agriculture, and chairman of the Mississippi Live Stock Sanitary Board, and Dr. G. B. Bradshaw, State Veterinarian, discussed the accomplishments of the Mississippi State Live Stock Sanitary Board and its plans.

The Association accepted the invitation of Dr. W. L. Gates to meet in Clarksdale in 1932. The following officers were elected for the coming year: Dr. R. H. Stewart, president, Dr. C. E. O'Neal, vice-president and Dr. G. B. Bradshaw, secretary.

HARTWELL ROBBINS, *Reporter.*

Vesicular Stomatitis Described in Bulletin

Although vesicular stomatitis, in horses, mules and cattle, is of rare occurrence in the United States, there have been three outbreaks in recent years—one in the East and one in the Middle West, in 1925, and one in the South, in 1929. The similarity of the early symptoms of the malady to those of foot-and-mouth disease sometimes gives veterinarians cause for alarm. A full description of the disease and its history is presented in Department Bulletin 662-D, "Vesicular Stomatitis of Horses and Cattle," just issued in revised form by the U. S. Department of Agriculture. The bulletin explains how to differentiate vesicular stomatitis from foot-and-mouth disease, and discusses the results of recent experiments with vesicular stomatitis and its treatment. Department Bulletin 662-D may be obtained from the Office of Information, U. S. Department of Agriculture, Washington, D. C.

The March issue of the JOURNAL will coniain the Proceedings of the thirty-fourth annual meeting of the U. S. Live Stock Sanitary Association, held in Chicago, December 3-4-5, 1930.

NECROLOGY

JOSEPH CHARLES FOY

Dr. Joseph C. Foy, of Centerville, S. Dak., died October 20, 1930. He was born at Vail, Iowa, May 23, 1881. At the age of 11 he removed, with his parents, to Centerville where he came to be one of the town's most respected citizens. Some time after his graduation from the Centerville High School, in 1899, he decided to study veterinary medicine at the Ontario Veterinary College. He was a member of the class of 1908. Dr. Foy never married. For many years he was an active member of the Centerville Volunteer Fire Department and served as secretary of the organization for a number of years. He was a member of the Knights of Columbus. Two brothers survive him.

JOHN A. LOWELL

Dr. John A. Lowell, of Shawnee, Okla., died in a local hospital, December 11, 1930, after a very brief illness. Death was due to a ruptured appendix.

Born at Bradford, Iowa, April 22, 1866, Dr. Lowell moved to Eureka, Kansas, with his parents, while still a boy. He attended local schools and then entered the Kansas City Veterinary College. He was graduated in 1910 and entered private practice at Yates Center, Kansas. Later he located at Ada, Oklahoma, and in 1915 moved to Shawnee, where he continued his practice until his fatal illness.

Dr. Lowell first joined the A. V. M. A. in 1917. He was active in the Oklahoma State Veterinary Medical Association. He was a member of the Christian Church and an Odd Fellow. He is survived by his widow, one son and one daughter.

C. H. F.

CHAUNCEY DEPEW MAULFAIR

Dr. Chauncey D. Maulfair, of Granville, Ill., died at the State Hospital, at Watertown, January 3, 1931. Pneumonia was the cause of death.

Born near McNabb, Illinois, October 7, 1876, Dr. Maulfair spent practically his entire life in Putnam County. He attended Granville High School and took a course at the Northern Indiana Business Institute, at Valparaiso, Indiana, and then entered the Chicago Veterinary College. Upon his graduation, in 1902, he entered private practice at McNabb, Ill., later locating at Granville. He was a former president of the village board of Granville and served a term as Sheriff of Putnam County.

Dr. Maulfair joined the A. V. M. A. in 1909. He is survived by his widow (née Minnie M. Sill), one son, two sisters and two brothers.

M. W. SHEMPF

Dr. M. W. Shempf, of Champaign, Ill., died suddenly January 15, 1931. About two weeks prior to his death, Dr. Shempf had his left leg amputated above the knee, following an injury inflicted by a horse some months previously.

Born near Assumption, Ill., April 7, 1879, Dr. Shempf obtained his veterinary education at the McKillip Veterinary College. Following his graduation in 1902, he practiced at Tuscola, Shelbyville and Pana, Ill., later going to Normal, where he remained until a few months ago. He is survived by his widow, one daughter, one sister and one brother.

IRA KEMP ATHERTON

Dr. Ira K. Atherton, of Hyattsville, Maryland, died at his home January 26, 1931. Although he had been suffering from high blood-pressure for several months, his death was very unexpected. He had attended a banquet the night before, at the Hyattsville Chamber of Commerce, of which he was a vice-president. It is believed that the excitement incident to the occasion overtaxed him.

Born at Bloomington, Illinois, September 26, 1871, the son of the late Dr. Onesimus G. Atherton, who died April 2, 1923, the younger Atherton attended local schools and took a business course before entering the New York College of Veterinary

Surgeons. He was graduated in 1893. Two years later Dr. Atherton entered the service of the Bureau of Animal Industry, at Boston, Mass., and was assigned to meat inspection. Later he was transferred to Chicago, on the same work. In 1906 he was promoted to inspector-in-charge at Peoria, Illinois, where he remained until 1913. He then took charge of meat inspection at Ottumwa, Iowa.

When foot-and-mouth disease broke out in 1914, Dr. Atherton was placed in charge of eradication work in northern Indiana. Later he was in charge of the work in Michigan, with headquarters at Niles. Following the close of this campaign, he was placed in charge of hog cholera control work in Clay County, Iowa. In March, 1917, he went to Maryland to take charge of hog cholera control in that state, in cooperation with the Extension Service of the Maryland College of Agriculture.

The views which Dr. Atherton held on the subject of hog cholera, its spread and methods of control, gained for him a nation-wide reputation. He frequently appeared in the programs of veterinary meetings and in December last read a paper before the U. S. Live Stock Sanitary Association, entitled "My Experience with Hog Cholera." Several years ago he started a series "Vest Pocket Essays," which materially added to his reputation, as they were published all over the country and read by layman and veterinarian alike.

Dr. Atherton joined the A. V. M. A. in 1897 and was quite proud of his long membership. Until his death he was the only member on the roll who joined in 1897. Dr. Atherton also was a member of the U. S. Live Stock Sanitary Association, the Maryland Stockmen's Association and the National Association of B. A. I. Veterinarians. He formerly served as a member of the Common Council of Hyattsville. He is survived by his widow, one son, one brother and two sisters.

PERSONALS

BIRTH

To Dr. and Mrs. C. F. Schlotthauer, of Rochester, Minn., a son, John Carl, October 6, 1930.

PERSONALS

Dr. Walter R. Anderson (Iowa '30) has removed from Worthington, Minn., to McCook, Nebr.

Dr. J. H. Cheney (K. S. A. C. '07) has removed from Great Bend, Kans., to Norwood, Colo.

Dr. C. E. Bassler (K. S. A. C. '07), formerly at Shawnee, Kans., is now located at Ainsworth, Iowa.

Dr. J. T. Traylor (K. C. V. C. '13), of Harlingen, Texas, is president of the Hygeia Milk Products Company.

Dr. M. C. Fitzwater (Gr. Rap. '06), formerly located at Bloomingdale, Mich., is now at Paw Paw, Mich.

Dr. C. J. Mulvey (McGill '94), of Mooers, N. Y., has been acting as assistant county veterinarian for Clinton County, the past year.

Dr. A. S. Schlingman (O. S. U. '11), of Detroit, Mich., is president of the Detroit local branch of the Society of American Bacteriologists.

Dr. William Stratmen (Chi. '09), of Danville, Ill., has resumed his practice after an absence from his office of more than a year, caused by illness.

Dr. J. R. Severin (K. C. V. C. '10), formerly with the United Serum Company, at Wichita, Kans., is now with the Royal Serum Co., Kansas City, Kans.

Dr. J. W. Berry (A. P. I. '23) has resigned his position with the Tennessee State Department of Agriculture and has resumed his practice at Pulaski, Tenn.

Dr. N. S. Mayo (Chi. '89), of Highland Park, Ill., accompanied by Mrs. Mayo, started on an automobile trip to Florida, about the middle of January.

Dr. L. E. Johnson (O. S. U. '30) has resigned his position with the Ohio State Division of Animal Industry and has located at Melvin, Ill., for general practice.

Dr. J. W. Griffith (Ont. '92), of Cedar Rapids, Iowa, was elected president of the Linn County (Iowa) Veterinary Association, at the annual meeting held in December.

Dr. George W. Gillie (O. S. U. '97), of Fort Wayne, Ind., who retired as sheriff of Allen County, December 31, has resumed practice as a member of the firm of Gillie & Wiles.

Dr. Ralph A. Dunn (Amer. '91), formerly of Beverly Hills, Calif., is now located in Santa Ana, Calif., where he recently completed the erection of a very nice small-animal hospital.

Dr. C. B. Denman (O. S. U. '09), of Newark, Ohio, is reported to be recovering from an operation performed in Water Reed General Hospital, Washington, D. C., the latter part of November.

Dr. C. B. Cain (Corn. '23), of A. & M. College, Miss., has accepted an appointment on the staff of the Rockefeller Institute for Medical Research, Princeton, N. J., effective February 1, 1931.

Dr. T. J. Leasure (K. S. A. C. '30), formerly of Solomon, Kans., is now located at 837 New Hampshire St., Lawrence, Kans., having taken over the practice of Dr. J. A. Bogue (K. S. A. C. '21).

Dr. Ward Giltner (Corn. '06), of Michigan State College, addressed the December meeting of the Detroit local branch of the Society of American Bacteriologists, on the subject of Bang disease.

Dr. Harvey W. Campbell (Colo. '22), formerly located at Santa Barbara, Calif., is now stationed at Petaluma, Calif,. where he is engaged in poultry disease control work for the State Department of Agriculture.

Dr. Wm. J. Pistor (Wash. '26) has severed his connection with the Rose City Veterinary Hospital, Portland, Ore., to accept a position on the staff of the Califorinia Department of Agriculture. He is now engaged in poultry disease control work at Petaluma, Calif.

Dr. Henry B. Hannum (U. P. '20), field veterinarian of the Pennsylvania Bureau of Animal Industry, was recently transferred from tuberculosis eradication to meat hygiene and is now stationed at Harrisburg, Pa. His home address is 2939 Canby St., Penbrook, Pa.

Dr. E. L. Krieger (Gr. Rap. '02), of Benton Harbor, Mich., recently returned to his home after an absence of about three weeks. Accompanied by Mrs. Krieger, he took an automobile trip through the southern part of the United States, covering more than 3800 miles on the trip.

Dr. E. E. Hatton (O. S. U. '17), of Orwell, Ohio, recently was robbed of his pocket-book, as well as his automobile containing about 200 dollars worth of drugs and instruments. Some days later the car was found wrecked and abandoned at Indian Springs, Md. The drugs and instruments were found intact but the car was badly damaged.

Dr. Chas. F. Runnels (O. S. U. '30), who was formerly associated with Dr. Frederick Priest (O. S. U. '95), of Newark, Ohio, is now engaged in general practice on his own account at 68 E. Locust St., Newark, Ohio. Dr. Runnels reports his poultry practice increasing right along. He has made over 15,000 tests for bacillary white diarrhea and expects to make about 4500 more in the near future. He has also vaccinated over 6000 fowls for chicken-pox.

A Group of Michigan Ladies

The above photograph was taken during the clinic at the Fair Grounds, Detroit, at the 1929 convention. How many will be at Kansas City in August?